BRAIN.

JANUARY, 1885.

Original Articles.

ON APHASIA.[1]

BY L. LICHTHEIM, M.D.,

Professor of Medicine in the University of Berne.

WHEN we read the recent controversies concerning the symptoms of Aphasia and their explanation, we might readily come to the conclusion, that there exist differences between the methods of investigation adopted by the chief authorities, and that it would be, therefore, desirable first to reach unanimity upon this fundamental question of method.

Happily such an opinion would be erroneous, and we can have no doubt as to the way to follow in our investigations, as there is no such divergence. The method which has hitherto yielded the results does not differ from those used in the natural sciences. Starting from the observation of facts, it culminates in the explanation of these facts. The correctness of our explanations must be subjected to the control of further observations. Precisely the same course is followed in experimental research, with the exception that, in our present subject, the experiments are not instituted at the will of the investigator, but are

[1 This paper has been translated by me from the German manuscript; but our limits required that the original should be condensed. The whole task was by no means an easy one; the careful revision bestowed by Professor Lichtheim on the proofs, and for which I beg to return him my best thanks, has, however, materially relieved me from the responsibility I had assumed.—A. DE W.]

supplied to him by nature, and that he thus depends for them upon a happy chance. The erection of the building can therefore proceed at a slow pace only, and must rise by degrees as the result of many toilers' work.

Nor do we meet with any divergence of opinion as to the end to be attained. Our task is to determine the connections and localisation of the paths of innervation subservient to language and its correlated functions. On the supposition of our having reached this end, we should then be able to determine the exact place of any solution of continuity in these paths, and account for its symptomatic manifestations with the same precision as we do for those of a motor or sensory paralysis depending on a lesion of the peripheral nerves.

Now, although all are agreed that we have by no means as yet reached this point, opinions differ as to how near we have arrived. We may, however, congratulate ourselves upon the simple fact of there being some agreement as to the fundamental meaning of disturbances of speech from cerebral causes. The amount of superstructure which will be raised on such a foundation must depend, in individual instances, upon the personal temperament of the architect. There is room both for the enthusiast and the sceptic, who both have their function to fulfil in the race for truth. The only necessary condition for the successful building up of the edifice, is that the one should not deny, the other not distort, acquired facts.

It follows from what I have already said, that every step which brings us nearer the fulfilment of the task before us must enable us to differentiate more accurately the clinical forms of aphasic disturbance. What to-day appears to us as a curiosity, as a case aberrant from the ordinary type, will to-morrow be classified as an instance of conformity to the law. This we shall find illustrated in the history of the previous researches into the nature of Aphasia. Broca was led, after many mistakes had been made, to bring into sharp relief aphasia in its narrower sense. Wernicke[1] was the first, to my knowledge, who distinguished between the latter symptom and those due to an interruption in the centripetal afferent paths. Besides these two chief forms, which he calls motor

[1] 'Der Aphasische Symptomencomplex;' Breslau, 1874.

and sensorial aphasia respectively, Wernicke describes a third, and designates it as commissural aphasia (*Leitungsaphasie*). The following discussion, the object of which is to establish a further differentiation, will bear upon this triple division of Wernicke's, and give me the opportunity of mentioning the other categories of aphasia. I abstain from any further reference to the historical aspect of the question, which has been treated by Kussmaul[1] in a way which leaves nothing to be desired.

The morbid types which I intend to discuss in the following remarks have been determined, in as far as they are new, deductively: it was the task of subsequent clinical observations to test the validity of the inferences. The necessity of differentiating still further the types of aphasia struck me on attempting to schematize the forms hitherto known, for the purposes of instruction. But I did not consider that my schema should be published until cases had been observed which coincided with the new types postulated therein. The important element of my task lies in the observations themselves and in their interpretation. Still I thought it advisable, in presenting my results, to follow the same path which I had myself trodden, giving in the first place the schematic representation from which I started in elaborating my views of aphasia. It will be seen that my conception is intimately connected with previous ones, especially with that of Wernicke: the points of difference will appear in the course of the argument.

The schema is founded upon the phenomena of the acquisition of language by imitation, as observed in the child, and upon the reflex arc which this process presupposes. The child becomes possessed, by this means, of auditory memories of words (auditory word-representations[2]) as well as of motor memories of co-ordinated movements (motor word-representations).[3] We may call "centre of auditory images" and "centre of motor images," respectively,[4] the parts of the brain where these memories are fixed. They are designated in the schema

[1] 'Die Störungen der Sprache;' Ziemssen's 'Cyclopedia,' vol. xiii. Leipzig, 1877.
[2] ['Wortklangsbilder;' or auditory word-impressions.]
[3] ['Wortbewegungsbilder;' or kinæsthetic word-impressions.]
[4] ['Klangbildercentrum;' and 'Bewegungsbildercentrum.']

by the letters A and M. The reflex arc consists in an afferent branch *a* A, which transmits the acoustic impressions to A; and an efferent branch M *m*, which conducts the impulses from M to the organs of speech; and is completed by the commissure binding together A and M.

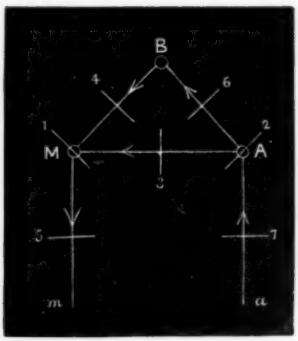


FIG. 1.

When intelligence of the imitated sounds is superimposed, a connection is established between the auditory centre A, and the part where concepts[1] are elaborated, B. All the diagrammatic representations of these phenomena agree so far, at least those given by German authors; beyond this, controverted points are found. The next step in the formation of volitional, or intelligent, speech involves a centrifugal connection between B and M. Our schema introduces, in consonance with most others, a direct commissure, whilst Kussmaul makes it pass from B through A to M. We shall soon return to this point. Leaving aside the point B for the present, we see from the diagram that seven different interruptions may occur in the paths therein assumed. It is manifest at first sight how variously the function of language may be disturbed by some of them; yet it is necessary, in order to gain a clear conception of the various types so produced, that we should include in our survey the disturbances of the functions of language involved in the acts of reading and writing. These are acquired in connection with the

[1 'Begriffe.']

exercise of speech, and are hence intimately connected with it; the same nervous paths are, to some extent, brought into play. Reading postulates the existence of visual memories of letters and of groups of letters. We may learn to understand writing through the connection between such visual representations (centre O) and auditory representations: by spelling aloud we bring the auditory centre into action, and thus establish a connection, through the path O A, between O and B; in reading aloud, the tract O A M *m* is thrown into activity.

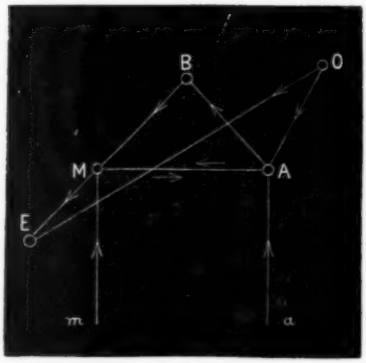


FIG. 2.

The problem with reference to writing is more complicated. The necessary movements have to be learnt, and associated with the visual representations; this is done through the commissure O E, designating by E the centre from which the organs of writing are innervated.

It is more difficult to determine the path through which volitional, or intelligent writing is executed. This tract must unite B with E, and clinical facts leave no doubt that it passes through M. There may be some doubt as to whether it leads directly hence to E, or passes round through A on its way thither. I shall return to this question presently; and adopt provisionally the former view, according to which Diagram 2 is constructed.

This figure makes it easy to derive the symptomatic type characteristic of each of the several possible interruptions in

the reflex arc. I have been in the habit of using it for several years past in my lectures, and have found that it greatly facilitates to beginners the mastering of an otherwise very complicated subject. But if the schema is to have any value beyond that of an aid to teaching, it must be shown that the seven derivable types do really correspond to existing forms of aphasia, and that clinical observations are fairly reducible to them. I shall begin my discussion with an *exposé* of the several types.

1. Interruptions in M—the centre of motor representations of words, or motor centre of speech—give rise to the following association of symptoms:

Loss of (*a*) volitional speech;
(*b*) repetition of words;
(*c*) reading aloud;
(*d*) volitional writing;
(*e*) writing to dictation.

There still exist:—
(*f*) understanding of spoken words;
(*g*) understanding of written words;
(*h*) faculty of copying.

This constitutes the true "aphasia" of Broca, as well as the "motor aphasia" of Wernicke, and the "ataxic aphasia" of Kussmaul. The interpretation usually given of this trouble of speech is the same as that involved in my plan. It is this aphasia which rests upon the firmest basis, and I need not adduce examples from the large number of those on record, such as, for instance, the two celebrated cases of Broca's. I believe I have seen some pure cases of this description, but I have no notes of them, and their investigation was not conducted with the fulness of detail necessary to clear up the only point among the symptoms which still remains to be elucidated. I am referring to the question, whether there is understanding of written language in such cases. According to the diagram, the power of reading aloud should be lost, but that of silent reading preserved intact. I am sure that in uncomplicated instances this must be so, but I much regret that I have not had such a case to observe during the last few years; for the study of the literature of the subject has not

afforded me any positive proof. In the majority of published cases no sufficient attention has been bestowed upon this question. It has indeed often been specified that the faculty of reading was intact: on the other hand, Trousseau[1] has shown that many 'aphasics' appear eager in their reading and yet do not understand what they read. He makes this observation precisely with reference to patients in whom the symptom of deficient understanding of writing was associated with the typical signs of Broca's aphasia. I shall return to this point, and show by personal observations how I think this contradiction may be explained.

2. If the continuity be broken at the point A, in the acoustic word-centre we find loss of:—

(*a*) understanding of spoken language;
(*b*) understanding of written language;
(*c*) faculty of repeating words;
(*d*) faculty of writing to dictation;
(*e*) faculty of reading aloud.

There is preservation of:

(*f*) faculty of writing;
(*g*) faculty of copying words;
(*h*) faculty of volitional speech.

This type corresponds with the "sensorial aphasia" of Wernicke, who has himself shown that though the faculty of volitional language is not lost, yet there are considerable disturbances in it. The latter is incorrect, inasmuch as wrong words are used, the words themselves are altered by the introduction of wrong syllables, occasionally to such an extent that the language becomes wholly unintelligible. This form has been called also "paraphasia." The explanation given by Wernicke of the fact, that in sensorial aphasia such disturbances occur in spite of the preservation of the tract for voluntary speech, appears at first sight rather forced. He assumes that the nervous influx descending along the path B M *m* sends a branch current to A, and that this *subconscious* innervation of the auditory memories of words secures the correct choice and expression of them; and that irregularities

[1] 'Clinique Médicale,' vol. ii.

occur as soon as the co-operation of these elements ceases to take place.

I accept this interpretation, but with a modification, namely, that the mere excitation of the auditory representation is not sufficient to secure correct speech, but that this representation must enter into relationship with the concept; that therefore the commissure A B must necessarily be intact for the same purpose. Paraphasia will be observed when in the arc B M A B, an interruption has occurred in such a way that language is not altogether arrested. The causes of such a modification will be pointed out presently.

It is by no means difficult to ascertain through self-observation, that such an innervation of the auditory word-centre

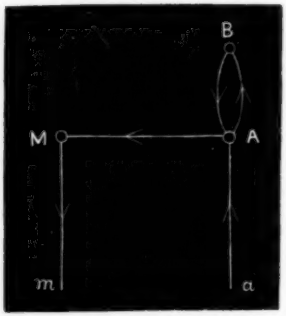


FIG. 3.

does really take place. When speaking aloud, we cannot control the fact, because the words are actually heard, and the innervation of A from *a* being much more powerful, conceals that of A from M. But if we perform only the movement of the mouth necessary to the emission of a monosyllable, without issuing it, we shall be most distinctly conscious of the corresponding auditory representation. This observation has perhaps given rise to the assumption of Kussmaul, that the path from the concept- to the movement-centre passes through the auditory centre. This is one of the points on which his schematic representation differs from the one given above. His diagram corresponds to the one given here (Fig. 3), and his path for voluntary language would be B A M *m*.

But it seems to me that this view does not correspond to the facts; the disagreement begins already in the case of Broca's aphasia, of which Kussmaul refers the seat, as we do, to the motor centre M. He assumes that the auditory representations are intact, and may be innervated from the concept-sphere; but this is, I believe, not the case. Trousseau, intending to prove that in aphasia words were forgotten, showed that in numerous cases the "inner speech" had disappeared. I have obtained evidence of this in a patient suffering from a form of the disease allied to Broca's type, and which will be adduced further on. It will generally be found a difficult task to elucidate this point satisfactorily; this is the method I use: I ask the patient to press my hand as often as there are syllables in the word to which an object corresponds. Those who have not lost the auditory representations can do this, even if their intelligence be limited, as I have been able to satisfy myself even under the least favourable circumstances. For instance, a patient who, besides a focal lesion of the right hemisphere, had had a hæmorrhage in the left half of the pons, and suffered, among other pseudo-bulbar symptoms, from complete speechlessness, preserved the faculty of fulfilling the test to the very last.

I have unfortunately, as was just said, not observed recently any pure cases of Broca's aphasia, but I have had a series of mixed cases in which this type predominated at least, and of pure cases of allied forms. I always found that the patients had lost the innervation of the auditory word-representations. These observations correspond so accurately to those of Trousseau, that I feel convinced that in ordinary cases of ataxic aphasia, the path from concept- to sound-centre must be interrupted.[1] Hence the diagram of Kussmaul cannot be correct, nor can a lesion in the path B A (Fig. 3) explain what occurs, for then the arc *a* A M *m*, and the power of repeating words would remain intact; whereas the latter, in Broca's aphasia, suffers as much as that of volitional language.

These considerations show that our schematic diagram may be considered as accurate, and that the co-operation of the

[1] A rare species of aphasia, included by Kussmaul under the ataxic, and to which we shall allude presently, differs in this particular.

auditory representations in speech is calculated to support the interpretation given by Wernicke of the paraphasic phenomena in his sensorial aphasia. I cannot refer to any typical instance of sensorial aphasia from my own experience; but Wernicke has given one,[1] carefully and minutely described, and in which the symptoms coincided[2] with the deductions from our schema.

3. The third form of aphasia, caused by the interruption of the commissure M A, has also been described by Wernicke, and corresponds with what we find in the diagram.

There are preserved:

(*a*) Understanding of spoken language;

(*b*) understanding of written language;

(*c*) copying words.

Paraphasia and paragraphia are present; showing, in accordance with what has been said previously, the existence of:

(*d*) volitional language; and

(*e*) volitional writing.

Similar disturbances are observed in the

(*f*) repetition of words;

(*g*) reading aloud;

(*h*) writing to dictation.

The path generally used in these acts (from A to M) is interrupted; still they are not completely in abeyance, because the tract A B M may be substituted for it. These actions, therefore, are not those of repeating, reading, and writing to dictation, properly so called, but are the result of impulses from the concept-centre; hence they manifest the same disturbances as volitional speech.

Cases of this sort do not appear to be very rare, though I am not acquainted with any observation of the kind as complete as could be wished. This is the more to be regretted, that it is precisely here that I have some doubts as to whether the construction of the path of volitional writing is correct. I have mentioned previously that I hold it as doubtful whether the path B M E be really used in volitional writing, or whether

[1] Loc. cit. p. 39.

[2] Excepting a difference in the power of volitional writing. This point will be considered in detail further on.

the channel of innervation be not from B through M and A to E (Fig. 4). The circumstance, that in volitional writing we are conscious of the excitation of auditory representations, does not prove that the *direct* innervation current really passes through the centre for the latter. The conditions are the same here as for the relation existing between these word-representations and volitional speech; whilst the chief current flows along B M directly to E, a secondary current may diverge from

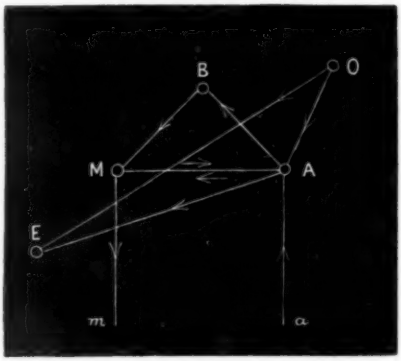


FIG. 4.

M through A to B, in order to secure the correctness of writing, as it does in the case of speaking. Clinical observations are necessary to decide on this point also; and the solution will be found in cases (described under 2) of Wernicke's sensorial aphasia, and of aphasia due to an interruption in the path M A. If localising the lesion of sensorial aphasia in A, we consider Diagram 4, the faculty of writing must have been lost too; whereas paragraphia only will be present, on the assumption that the relation of parts is as in Figure 2.

Though the question, when framed in these terms, appears a simple one, yet it is far from being easy to find an answer to it, because the published material at our disposal scarcely contains any sufficiently precise data to give us certainty on this point. The only case which we may adduce is the one already quoted of Wernicke's;[1] the patient could not

[1] Loc. cit. p. 39.

write: "When sitting at the table to write, she takes the pencil presented to her point upwards, looks at it, turns it round, holds it properly, but makes only up and downwards strokes. When the pen is given to her in the same way, she turns it round, dips it properly into the inkstand, holds it properly, but with no better result." The agraphia persisted throughout, whilst the disturbance in the expression and intelligence of speech had disappeared. This case points to the correctness of Diagram 4; but it is not safe to draw a definite conclusion from a single instance, and unfortunately all other published cases of word-deafness fail us here, as will be seen on reading the thesis of Skwortzoff.[1]

Precisely in the same way the form of aphasia arising from an interruption in the path M A, ought, according to Diagram 4, to be accompanied with a loss of volitional writing; whilst, according to Diagram 2, paragraphia ought to be present: but we cannot decide with absolute certainty from the observations at our disposal. In both of Wernicke's cases agraphia was present; whilst others, scarcely reported with sufficient accuracy however, leave room for doubts. In a case of my own the clinical aspects clearly corresponded to this type, whilst the post-mortem appearances coincided with Wernicke's description. I relate it here, because such autopsies are rare; but, owing to the insufficiency of the notes, it cannot serve in deciding on the point we are discussing.

[1] 'De la cécité et de la surdité des mots dans l'Aphasie,' Paris, 1881. On page 84 a Case of Broadbent is mentioned which could be opposed to that of Wernicke. It is one apparently of sensorial aphasia, for besides imperfect understanding of language, the speech consisted in a kind of unarticulated jargon. It is said that the writing was not involved. But apart from the fact, that it is doubtful whether this was really volitional writing, and not only that to dictation or to a copy, the whole description is given in such a fragmentary manner, that we cannot tell whether we have to do with true sensorial aphasia, or some variety of it. The case, therefore, cannot be brought as evidence on the subject before us.—Since this paper was in the press, I read a typical case of Wernicke's sensorial aphasia described by Grasset (Contribution clinique à l'étude des Aphasies. 'Montpellier médical,' Jan. 1884) and in which also there was complete agraphia. This case too, therefore, points to the correctness of Fig. 4.

CASE I.—*Right hemiplegia; aphasic disturbances.—Obliteration of the Sylvian artery; brown softening of the left insula and neighbouring portions of frontal and temporal convolutions.—Pachymeningitis interna.—Valvular heart-disease, &c.*

J. S. B., aged 46, a labourer in Thun, was brought into hospital at Berne on the 3rd of April, 1883. We failed to obtain any clue to the beginning and course of the disease.

Sensorium normal. *Speech* much altered. When asked to relate his history, he strings together in a fluent manner numerous words, of which scarcely one now and then can be made out. The following were noted: "Evening, five and twenty, and." Patient is aware of the incorrectness of his diction, and tries to assist himself by gestures. He succeeds better with single short words and answers; thus, in answer to the question, "What was there for supper?" he answered, "Bread, meat, potatoes," with only two mistakes. His own name he mutilates.

Repeating.—When he repeats connected sentences he manifests the same defects as in volitional speech; single short words are pretty correctly rendered. No note taken of the way the patient names objects.

Understanding of Speech entire.—*Understanding of Writing*, whether printed or hand-written, is preserved. On reading aloud he makes the same mistakes as in talking.

Writing very imperfect. He mixes up the order of the letters in a word; and usually stops after an attempt of short duration. He can copy what is set before him with absolute correctness.

Motility.—Slight paralysis of the right arm and leg; apparent on simultaneous action of both sides of the body. Paresis in buccal region of right facial.

Sensation normal. The tongue deviates to the right, and is the seat of tremors. We pass over the symptoms in the chest and other organs. Symptoms remained unaltered until the beginning of May. Then consciousness became obscured during the next few days, and he died in the night of the 8th to 9th of May.

The autopsy was made, and a full report drawn up, by Professor Langhans. The brain presented the following appearances:

Inflammatory membranous deposits on the inner surface of the dura-mater on the left side. Pia-mater somewhat opaque and whitish, especially over ascending, and apex of temporal convolutions, where it is adherent.

A good deal of serum at the base of the brain There is a

considerable depression of the convolutions bordering the upper and posterior part of the left Sylvian fissure, as well as of the ascending frontal and parietal convolutions. The Island of Reil is sunken, forming a depression into which the second frontal convolution falls suddenly to a depth of 1½ ctm.; the third (lowest) frontal convolution terminates into it likewise. Here the pia is inflamed with yellow discoloration. The depression is bounded posteriorly by the fissure of Sylvius. The middle portion of the first temporal convolution is somewhat sunken opposite the depression of the Insula, in which the consistence of the cerebral matter is soft, with harder patches around.

It is difficult to follow the Sylvian artery beyond the point where it reaches the Insula, and presents on the length of 1½ ctm. a tough whitish appearance; it is occluded just before the division and a short way up the posterior branch.

The softened patch occupies the bottom of the Sylvian fissure and extends to about 1½ ctm. of the posterior part of the inferior frontal convolution, and to the neighbouring portions of the ascending frontal convolution. A fragment of the cortex of the ascending temporal is also wanting, but there is no yellow discoloration here.

The case just described is undoubtedly one of the third kind of aphasia; the symptoms tally with the theoretical postulates established previously. But the point which to us is most interesting, the capacity of the patient for volitional writing, is not established in the notes, because it is not sufficiently distinguished from writing to dictation.

In the absence of more definite data than those already adduced, whereby to decide which course the impulses follow in volitional writing, it appears to me more probable that Diagram 4 gives the more correct construction. There is one difficulty, however, and it is that in Broca's aphasia, writing to dictation is disturbed, a fact not deducible from Diagram 4, but with which Diagram 2 appears more consonant. On the other hand, as we shall see, in some cases where volitional speech is lost, but not the faculty of repeating words, the first syllables only are repeated correctly; when the patient is to repeat a sentence it is necessary to say it word by word. The explanation of this phenomenon is easy. If four or five words are given together, the patient must retain them in his

memory, and thus what he says ceases to be a repetition, properly so called, but is an effect of volition. This is still more the case with reference to writing, an act which takes a much longer time; but few letters are written really to dictation, and indeed one finds in many cases of Broca's aphasia that the first letter only of a word can be written down, the rest of the word being unreadable.

Further observations cannot fail to throw light on the question. This controverted point does not apply to the remaining forms of aphasia.

4. A variety of motor aphasia is created by interruption of the path B M, of which we have many examples. From the diagram we should expect the loss of

(*a*) volitional speech;

(*b*) volitional writing;

whilst there are preserved—

(*c*) understanding of spoken language;

(*d*) understanding of written language;

(*e*) the faculty of copying.

So far the symptoms coincide with those of Broca's aphasia. They differ inasmuch as there is preservation of

(*f*) faculty of repeating words;

(*g*) writing to dictation;

(*h*) reading aloud.

As an example of this kind of aphasia, characterised by loss of volitional speech and preservation of power of repeating words, I may mention a case of Hammond.[1]

Still more striking is the faculty of reading aloud. Most recorded cases are incomplete in this respect. I therefore give a case of my own which beautifully illustrates this point.

Case II.—*Traumatic aphasia.*

Dr. C. K., a busy medical practitioner, had a carriage accident, and was carried home unconscious. Three hours afterwards he was bled, when consciousness returned. There were severe bruises on the right side of the body, and of the head, which was ecchymosed. The movements of the right arm and leg were never

[1] 'Diseases of the Nervous System,' 7th ed., chap. vii.

absent, but weakened, whether through paralysis or injury is not certain. Immediately after venesection the right hand could be stretched firmly, only lifting it was difficult. Sensation normal. Difficulty of swallowing during the first two days; fluids readily got into the air-passages; mouth not easily opened; tongue difficult to pull out. Patient got up after about a week, when it was noticed that he dragged the right leg and swayed a little.

Speech was much affected: the first day he only said "yes" or "no," but quite appositely. Gradually, more and more words returned, at first imperfect. Whilst his vocabulary was still very meagre, it was observed that he could repeat everything perfectly. Soon after the accident he began to read with perfect understanding. It was established beyond doubt that he could *read aloud* perfectly at a time when he could scarcely speak at all. The statements of his wife are most positive and trustworthy on this point, though he himself does not remember what took place just after the accident. She states that after much difficulty in making himself understood by gestures, he obtained a newspaper, and to the great astonishment of all present he began to read fluently. She herself thought it most strange and inexplicable.

He could not write voluntarily at all; but this faculty returned slowly and imperfectly, as did speech. On the other hand, he could, soon after he had left his bed, *copy and write to dictation*. He spoke German and French fluently before the injury; but German rather the better of the two. As the aphasia diminished, German words returned before French.

Immediately after the accident the right pupil was dilated and immovable; no troubles of vision. I saw the patient six weeks afterwards, at a time when the loss of speech had to a great extent disappeared. One could observe a slight paresis in the right lower facial region; some weakness of the right leg, but nothing noticeable in the right arm. Sensation was normal, except that he did not so readily recognise objects with his right as with his left hand. The right pupil sluggish; no disturbance of vision nor hemiopia.

In every other respect he is in a normal condition, with the exception of speech. His vocabulary is copious, but he does not talk much, and speaks in a drawling manner. From time to time he misses a word or construction; he then tries to express himself with gestures. Speech much more defective when he must name objects shown to him; then many names escape him, and he also makes mistakes (*e. g.* for "Bild," he says "Milbe," then corrects himself and says "Portrait"; for "Stahlfeder" he says first "Bleifeder," then "Tintenfeder"). Many words are missed in

French also; he finds the French equivalents of the words he can say in German. Patient says himself that the auditory representations of the words he cannot find are missing; he cannot tell the number of syllables in them. If these words are spoken to him or written before him he says them at once, but forgets them immediately. He repeats correctly whole sentences, if not too long.

He understands spoken and written language perfectly. He can read aloud with the greatest fluency, and with scarcely any stoppage. Writing very imperfect. Asked to write the history of his illness, he puts down mutilated, meaningless words, in which it is impossible to discover any sense. Writing single words, names of objects shown, gives a somewhat better result. There is a parallelism here between writing and speech; such names as he cannot say, neither can he write.

On dictation, he writes fluently and without faults. The strokes are somewhat clumsy. He copies equally well. Intelligence normal. His wife assures me that he manages business perfectly, thinks of things in time, &c.

I saw the patient again a month later. Great improvement in his speech, which is fluent, and with almost no hesitation; he names objects with much less difficulty; words are rarely mutilated. Writing is still deficient, though much improved. What he writes is perfectly legible.

5. Still better known than the preceding is the type of aphasia arising in an interruption of the path M *m*. The diagram indicates loss of:—

(*a*) Volitional speech;
(*b*) repetition of words;
(*c*) reading aloud.

Preservation of:—

(*d*) Understanding of speech;
(*e*) understanding of writing;
(*f*) faculty of copying.

Again, and as a distinguishing feature between this and Broca's type, there remain:—

(*g*) Faculty of volitional writing;
(*h*) of writing to dictation.

There are numerous examples of patients who had lost the faculty of speaking, and could yet make themselves understood by writing. I have seen several cases of this description,

and possess the notes of one in which the patient was under my care several years ago, and wrote his history himself. But it would be useless to relate it here, in presence of the material already at hand.

According to Kussmaul, such cases present the true uncomplicated type of ataxic aphasia, that is, of that form of speechlessness, "in which patients, with freedom of intellect and of movement of the tongue, have the memory of words as acoustic signs, but are unable to emit them. The proof that they really possess these signs is found in the fact, that they can embody them in writing. When asked to articulate them as sounds or words, they cannot, even if they are shown how to shape their tongue and lips."

It is obvious that this description includes the type now before us, and Kussmaul gives a series of most pregnant instances in his chapter on ataxic aphasia. But the aphasia he so designates extends over a wider range of cases, such as those of Broca's aphasia, which we have discussed under our first heading. In these cases there is ataxic agraphia also, owing to the implication of the centre of co-ordination for written signs, which is distinct from, though connected with, that for spoken signs. The close relationship between these centres explains the fact, that the two symptoms so often co-exist.

Kussmaul's views rest on the assumption that, as the motor tract of spoken language is innervated by the left hemisphere only, so is the tract of written language; for it is only under such circumstances that a lesion of the left hemisphere could cause agraphia by an injury to the writing centre. I cannot accept this interpretation, but hold it more probable that the innervation of the movements of writing originates in both cerebral hemispheres.

When we learn how to write, we apparently employ the left hemisphere only; but we must assume that the right hemisphere is the seat of slight nervous action as well, for it can be shown that a certain amount of facility is also imparted for the same movements with the left hand. When the left hand executes "mirror-writing," it performs the same movements as the right hand in ordinary writing, and it is easy to

show that the left hand writes mirror-wise better than it does the usual type. In my own case, even, the strokes are more correct if I write in English characters. Now it could be objected that this innervation of the left hand flows from the left hemisphere; but this is improbable, for in some lesions of the left hemisphere it happens that this tendency of the left hand to execute mirror-writing becomes manifest. Moreover, the fact, that in complete right hemiplegia the power to write with the left hand is not diminished, is opposed to the unilateral innervation of the movements of writing. Therefore the point E, which in our diagram represents the centre of writing movements, must not be looked upon as single; in addition to the point E in the left hemisphere, there is a corresponding spot, E′ in the right, to which commissures go, and which can

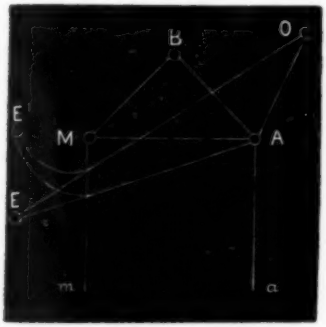


FIG. 5.

come into action when there is some lesion of E. There are other proofs that in agraphia the motor innervation of the movements of writing is not involved. In pure cases of Broca's aphasia the faculty of copying is preserved,[1] whilst that of reading aloud, and of every kind of repetition, is lost. On the other hand, these patients are not only unable to write, but cannot even compose words with letters placed before them. The real cause of the agraphia does not depend on any lesion of the motor apparatus, but on the impossibility of

[1] See example given by Wernicke, loc. cit., p. 56.

reproducing voluntarily the auditory representations.[1] I have already shown how one can convince oneself that such patients are wanting in the faculty of volitional recall of the auditory representations, and have stated how far I had reached certainty on this point.

Since the above has been written, a case of Pitres[2] has been published, which gives direct evidence of the correctness of my views on agraphia. The patient was suffering from agraphia, the residual sequel of an apoplectic seizure; speech, understanding of language and writing were intact, whilst volitional writing and writing to dictation were impossible, the faculty of copying being preserved. The lesion would be referred by me in the path A E (Fig. 4). The interesting point about the case is, however, that this agraphia affected solely the right hand; the patient could write correctly with the left hand, and copy with his right what he had written with his left hand. This shows the correctness of the diagram, in which the centre E is represented as existing in both hemispheres, and the path A E as being double.

It is obvious that this agraphia has nothing in common with that occurring in Broca's aphasia. As could be expected, the patient could make up words from their component letters, a feat impossible to those suffering from Broca's aphasia.

Pitres adduces two more cases of isolated agraphia. The first, quoted from Ogle, is apparently much more complex. The patient could not even copy, nor make up his name with the letters, and had serious embarrassment of speech. The lesion was probably a more extensive one, and took its origin in an injury received eight years previously. In the second case (from Charcot) the conditions were likewise less simple. We are not told whether the agraphia was bilateral. I may add, that the paths A E and A C may be interrupted at the same time, as they appear (see Fig. 5) to run together in the brain for some distance before they separate to go into the two

[1] We learn to write with the co-operation of auditory representations, hence, we cannot write without it. Every lesion of the path B M A (Fig.) 4 destroys the innervation of the auditory representation from the concept-centre, and must necessarily result in agraphia.

[2] "Considérations sur l'agraphie à propos d'une observation nouvelle d'agraphie."—'Revue de Médecine,' 1884, 11.

hemispheres. Isolated agraphia will therefore, under certain circumstances, appear as bilateral.

The theoretical types with which we have hitherto been engaged corresponded closely with well-known varieties of aphasia. There remain two, due to lesions of the commissures A B and A *a*, which must present themselves as cases of sensorial aphasia. Owing to the rarity of examples of the latter, it would have been but natural had I had no actual examples of it to adduce, though it would obviously have weakened my whole position, the full confirmation of which requires a filling up of every gap. I have, however, found the two missing types, and their entire correspondence with the theory of aphasia here developed is a warrant for the accuracy of the hypothesis.

6. Lesions of the path A B, would give rise to the following symptoms. Loss of:

(*a*) understanding of spoken language;

(*b*) understanding of written language.

There are preserved:

(*c*) volitional speech.

For the reasons stated above, there will be paraphasia. So far, the symptoms coincide with those of Wernicke's sensorial aphasia. Here, however, there subsist,

(*d*) volitional writing,

which will also have the characteristics of paragraphia;

(*e*) faculty of repeating words;

(*f*) of reading aloud;

(*g*) of writing to dictation.

Owing to the interruption of communication between A and B, there must be a complete loss of intelligence for what is repeated, read aloud, written to dictation by the patient.

There also subsists the

(*h*) power of copying words.

I shall now relate a case of my own which presents all these characteristics.

CASE III.—*Aortic insufficiency. Cerebral softening. Sensorial aphasia.*

J. U. Schwarz was admitted into the Inselspital on the 19th of May, 1884. Sixty years of age—nothing noteworthy in antecedents.

He was well up to the 15th of May. The next night his wife noticed that he became restless, spoke in his sleep, rose and returned to bed, &c. At 4 in the morning it became apparent that something had happened; what he said had no sense. He did not answer to questions; he tried very hard to make himself understood, and the same mutilated expressions were constantly repeated. His wife thought he had lost his reason. His apparent intention was to complain of severe pains in the forehead and nape of neck. This state of things went on, and as he was losing strength, he was taken to the hospital.

Patient is a rather fat, pale, man. Features flabby; expression dull. On admission, he gave the impression of a subject with psychical deficiencies. He talked a good deal, but repeated the same phrases. He points to his head and says, "Oh how stupid I am, I cannot," &c. It is difficult to make anything out of him, for he understands little that is said to him; but it is easy to ascertain that he is in possession of his intelligence, and that the apparent psychical deficiency consists only in his difficulty in understanding others, and in making himself understood. His actions leave no doubt on this point.

Spoken language.—The disturbance in his power to understand it is very evident (verbal deafness). If one stands behind him and talks to him, he turns round and asks: "Do you speak to me?" The simplest requests, to show his tongue, shut his eyes, are answered by: "I don't know what one wants."

Speech.—There is no deficiency in his vocabulary. He talks a good deal in a flowing manner: he seldom is short for a word; he occasionally uses a wrong or a mutilated one. But this is unfrequent, and in this respect he has greatly improved on what his wife described he was. He is in great difficulty when he has to name objects shown to him; he finds the names with the greatest difficulty, and assists himself with descriptions. Instead of "wine," he says "that is strong;" for "water," "that is weak," &c.

Repetition.—He obviously could repeat correctly all that was spoken before him; but he apparently did not understand what he did repeat, as for instance the words, "My name is Peter Schwarz,

and I am already 4 years old," which did not draw any signs of denegation on his part.

Written Language.—He understands nothing printed, or hand-written. The simplest things placed before him he cannot read nor decipher. He knows the name of most letters, and gives it correctly; he only confounds the capital I.

Reading aloud.—He can make up letters into words, and he can read aloud by spelling; but it is evident that the sense of the word remains closed to him. Out of the sentence, "Will you have a glass of wine?" he goes correctly as far as "you" but has manifestly no idea of the meaning.

Writing.—His volitional writing is worse than his speaking—but he does write a few words correctly. In "Meine Frau" (my wife), he manages the first word, not the second.

Writing to dictation was not tested at the very beginning. Later it was found that he could do it well if each word was given singly; whole sentences are rendered inaccurately, words being missed.

Copying.—He copies perfectly, changing German into English characters, but does not understand anything of what he so writes.

Motility and Sensibility.—No difficulty in walking or standing. Movements of arms and legs quite normal; sensation likewise.

Reflexes.—Skin reflexes normal. Patellar and Achillis-tendon jerk absent.

Cerebral nerves.—1. Smell normal.—2. Nothing noteworthy.—3, 4, 6. Movements of the eye normal. Pupils small, react to light.—5. Normal.—7. No weakness of facial muscles.—8. Hearing not very acute; but present on both sides; he hears the tick of the watch only when close to the ear.—9. Taste appears to be normal. He can recognise and name acid; seems to recognise sweet, but cannot name it. He calls bitter "strong." Deglutition normal. 10. Voice natural.—11. Movements of the tongue free.

In the further evolution of the case, the word-deafness receded without disappearing altogether; on the 21st of May, the improvement was already very marked. On the 22nd the following conversation was held. "Shut your eyes."—He does it. "Take this glass of wine from the table."—He turns round and says: "Are you saying anything to me? I don't understand it." "Do you like red or white wine best?" "Here is some red wine." "Put your right leg out of the bed." "What shall I take?"—and he puts out the left. "Put your right forefinger to your nose."—"Some thing right? What must I do?" He puts the right leg out of bed. "Drink the Professor's health with this wine."—"As you wish. . ." The request is repeated. "Shall I take red wine for me or for you?" "Show your hand."—He does it. "Do you cough

much?"—"Only to-day, as the weather is so bad." "What costs the cheapest wine you buy?"—"I don't know the words any more," &c.

His writing improves also. On the 21st he could write the alphabet easily. German and English characters are used promiscuously. On the 29th he made the first attempt, on being asked, to write a letter.

"*Ich soll*[1] Herrn Professor etwas wissen, *aber was soll ich etwas gheides wissen.* Ich weiss garnichts davon."

He still fails to make out written language; but this does not hold for numbers. He reads the multiplication table, and tells the results, though with a little uncertainty.

At the beginning of June he understands what is said to him much better; his speech is to a slight degree paraphasic. But one notices now that conversation is easier, that he has difficulty in finding the names of objects shown to him, and that he immediately forgets them when they are told. Thus, he was shown a knife and asked "What is this?"—"I have seen something like it, but I can't remember the name." "Is it a knife?"—"Yes, knife, knife!" "What is this?" showing him a cap. He mutters, but cannot find the word. "Is it a coat?"—"No," pointing to his own coat. "Is it a cap?"—"Yes, a cap." "Tell us what you have been eating."—"Eaten something, this morning at 8 o'clock drank something, and again at 12 o'clock, but I can't tell more." "Take your pocket-handkerchief, and wipe your spectacles." Patient does it. "Take my purse, take five francs out of it, and give them to So-and-so." He takes the purse, and five francs out of it, but does not understand the rest.

His understanding of written language is very bad still, though when he reads aloud he makes out the meaning better, a fact which corresponds with his improvement as to spoken language. When he is not allowed to read aloud, he apparently spells inwardly.[2] Even what he copies to dictation is obscure to him. I dictated to him an I. O. U. for 20,000 francs, which he wrote down, and allowed me to put in my pocket without giving the least sign of emotion. Volitional writing is still accompanied with well-marked paragraphia, though there is improvement in this respect. He wrote the following letter to me on the 4th of June:—

[1] [The words in *italics* were written in English characters, the rest in German. The word "wissen" is apparently used for "schreiben." "Gheides" is meaningless.]

[2] When questions were asked, he, at the beginning of his stay in the hospital, used to repeat them, it seemed to facilitate his comprehension; but occasionally did not understand them even after repeating them.

"Juni Mittwoch d. 4ten.—Herr Professor Dr. Lichtheim. Ich möchte heute etwas schreiben. Ich weiss noch sehr wenig. Ich bin Arbergergasse No. 52, mit meiner meiner Lieben um 1 und ½ Stunde sehr gern gesehen. Ach wie tausend Mal so gern für seine liebes Herz. Ach gott wie gern sollte ich viel mehr wissen und mein liebes Herz müsste mal allte Tag, oft die weiss täglich hundert mal und ich weiss fasst noch nichts mehr was ich zu wissen sollte. O wie viel zu wenig daheim. Ich will am morgen Mittags daheim Arbergergasse zu meine Liebenfrau. Fünf mal heute noch. Ich will auch noch etwas schreiben.—Schwarz-Beer."

Patient left the hospital on the 6th of June, but came to see us once every week. Every time he showed further improvement. On the 23rd the following notes were taken:

1. *Volitional speech.*—Paraphasia is very slight; word-amnesia, on the other hand, is considerable, though not so apparent in current conversation. He remembers the names of objects a little better; still he is very deficient in this respect.

2. *Volitional writing* is improved. There is less paragraphia.

3. *Understanding of spoken language* is much better. Patient understands almost everything he is told, even the longer sentences. Complicated tasks are executed correctly. Still, beyond a certain point, he still manifests deficiency in this respect; and it is impossible to appeal to his self-observation.

4. He *repeats words* correctly, and in longer sentences. He does it more quickly and intelligibly.

5. *Reading aloud.*—More ready; but he still mainly does it by spelling. He understands better.

6. *Understanding of writing* more deficient than that of speech, though unquestionably improved. He understands short words at once. Longer ones take him an interval, during which he seems to put them together; he used to repeat them, but this being forbidden he appears to spell them inwardly.

7. *Copying and writing to dictation* are carried out properly. He understands much of what he writes. The experiment with the I. O. U. does not succeed any longer.

The last time the tests were applied was on the 25th of July. He still talks somewhat slowly, the arrangement of words is often peculiar, but it takes time to discover that words are wanting in his vocabulary. He is easily stopped by proper names, and finds them with difficulty. The word-amnesia, however, is much less marked.

He seems to understand all that is said; one is occasionally obliged to repeat a question; and he himself repeats, as if with

astonishment, proper names. Much paragraphia still present when he writes of his own accord, though a letter he wrote to me on that day shows a marked improvement. Progress is also noticed in the other respects.

On the 30th of July I was called to see him. About midnight his wife noticed that he suddenly became restless. He tried to get up, and fell. The left arm appeared to be paralysed; but he can give no account of the state of the sensorium. He has been restless since, and talked nonsense.

I found his consciousness slightly affected; he is continually babbling as a delirious fever-patient. He reacts a little when called, but does not recognise people about him.

The eyes are chiefly turned to the right; it is difficult to make him fix them; when called he answers "yes," but turns them more to the right, and does not follow the finger. Pupils rather contracted.

He appears to have headache on the right side, points to it with his hand, and applies a sponge to it. Left arm flaccid and paralysed; left leg apparently sound, yet on rising he bends towards the left side.

Left naso-labial fold somewhat effaced. Sensation and reflexes on the left apparently diminished. No paraphasia noticeable in what he talks. There was constant vomiting; and the patient was taken to another hospital, owing to my removal into other wards, and is still living.

A few remarks on this case, in which the symptoms agree with those postulated by an interruption in the commissure A B, may not be out of place. It may be asked why he spelt the words, though the path O A M *m* was intact. I think this is explained by the fact, that for fluent reading it is necessary to understand the words; we guess the words before we have actually read them through, and gain much time thereby. When we have to say long words in a foreign language, we also must spell to a certain extent. Our patient shows also a phenomenon which has been observed in other cases of the kind; he repeated the question of which he did not understand the meaning. This condition is closely allied to the *Echolalia* of Romberg; and Spamer,[1] who also deduced the possibility of this symptom from his schema, used this name. In our case it did not appear as if the repetition necessitated the under-

[1] 'Ueber Aphasie und Asymbolie;' 'Archiv für Psychiatrie,' vi. 523.

standing of the words heard, by means of a centripetal path[1] from M to B. The circumstance that the patient supplied his lost understanding of written language by reading aloud, must be explained differently. The case shows, with many others, that disturbances in the use of written language are much slower to disappear than those connected with speech. Paragraphia here persisted much longer than paraphasia, and the patient understood writing but very imperfectly, whilst verbal deafness had practically passed away.

This may be explained by assuming, that the recovery of the understanding of words is not effected by a new connection between A and B, but that the sound-representations are deposited at another point A′ which is connected with M, B, and *a*. We should thus obtain a state of things as represented in

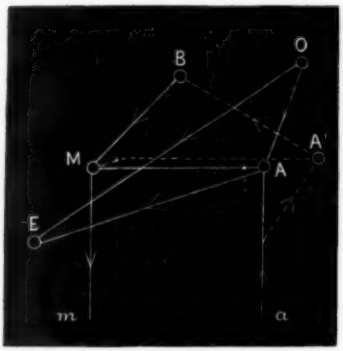


FIG. 6.

Diagram 6, where the destroyed path A B is not shown, but where the vicarious connections are depicted by dotted lines. With the assistance of the figure, one easily sees that the patient can easily understand by the ear what he reads aloud; whilst silent reading remains senseless. Silent spelling is already of assistance under these circumstances, because along

[1] I should not have mentioned this possibility, had not a case of Westphal's (cf. Spamer, p. 541) made such an interpretation probable.

[2] The rapidity with which this occurs may seem an objection to the explanation. But the sensorial aphasia of Wernicke through lesions of A goes on diminishing likewise, a fact not susceptible of another interpretation.

with the innervation of the movement-representations the words are revived in A′ and make understanding possible; The commissure O A′ and A′ E, require to be used for a much longer time, before the disturbance in the writing can be overcome by their substitution.

The case I have just related is by no means an isolated one, but appears to be of the same category as that of Schmidt,[1] and that of Broadbent[2] mentioned by Skwortzoff as one of word-blindness. In other instances, the observations are not sufficiently full to allow us to decide whether the cases really belong to the same group.

7. The last interruption to be considered, that in the path A *a* does not really belong to the subject of aphasia, for the faculty of speech remains perfect. Still it is necessary to consider it here, because its symptoms cannot be properly understood except in connection with the present subject.

From the diagram we should conclude that there is a loss of:

(*a*) understanding of language;
(*b*) faculty of repeating;
(*c*) faculty of writing to dictation.

The following are preserved intact:

(*d*) volitional speech;
(*e*) volitional writing;
(*f*) understanding of writing;
(*g*) reading aloud;
(*h*) copying words.

There is no paraphasia nor paragraphia, because the path B M A B is whole; we could therefore designate such cases as "Isolated speech-deafness," as the incapacity for repetition and writing to dictation may be included under the term "speech deafness." But since there is a form of speech-deafness, in which, as we have seen, the faculty of repeating and writing to dictation is preserved, it is advisable to distinguish sharply these functions.

[1] 'Allg. Zeitschrift für Psychiatrie,' 1871, vol. 27, p, 304; quoted by Kussmaul, p. 176.

[2] "Cerebral Mechanism of Thought and Speech." 'Med. Chir. Transactions, 1872,' vol. lv.; quoted after Skwortzoff, loc. cit., p. 38.

I have a personal observation to record of such a case which has already been published by Burckhardt.[1]

CASE IV.—*Chronic bronchitis. — Arterio-sclerosis. — Two apoplectic seizures; after the first, paraphasia and paragraphia gradually receding; after the second, persistent word-deafness.—Incapacity of repeating and of writing to dictation.—No disturbance of speech.*

Mr. L., aged 55, formerly a teacher and journalist, remained healthy till 1877. He then had an apoplectic fit, which was followed by troubles of language only, with the exception of a slight weakness in the muscles of the mouth on the left side of the face. These are fully described in Burckhardt's observation, to which the reader is referred for particulars. They consisted in paraphasia during volitional speech, repetition of words, and reading; paragraphia in writing to dictation. Volitional writing was very faulty, but not abolished. Nothing is said of the intelligence of language; but from the context and information given by his wife, it appears to have been intact. Evidence as to his understanding written language is wanting.

This association of symptoms may be identified with the one described under 3, as due to a break in M A, and the case is one I had in view when I said that the conditions of voluntary writing agree best with Diagram 2.

The state just described gradually improved, and a slight defect of speech only remained, consisting in the occasional misuse of a word.

In June 1882 he had a sudden attack; the symptoms seem to have been slight. A medical examination made immediately after showed an increase in the facial paralysis; and his wife said the defect of speech was more accentuated; he could not read nor write. We have here a discrepancy between the information given by the wife and that furnished by Burckhardt, who does not mention any disturbance of speech, and says he could read and write. I have mentioned the former statements because they do not agree so well with my views; but as they were made long after the event they do not deserve the same confidence as the notes taken at the time by Burckhardt. He was completely word-deaf.

The disturbance of language rapidly improved, even beyond the state in which he was before the attack. He recovered reading

[1] "Ein Fall von Worttaubheit."—'Correspondenzblatt für Schweizer Aerzte,' 1882, No. 20.

and writing completely; but the word-deafness remained the same. Intelligence free: patient felt weak, and did not like to leave his bed; he suffers from chronic catarrh and asthma.

I saw him first in July 1883; and again in June 1884; on both occasions his condition was the same.

[Passing over the general examination of the body, which presented no features of importance, we come to the functions of language.]

Understanding of speech.—Patient gives the impression of an absolutely deaf man, and differs in this respect from other cases of word-deafness. One cannot have the least communication with him except through writing; even his wife cannot make herself understood otherwise, as he can read from the lips but very few words, and these imperfectly.

I should probably have taken him for really deaf had I not been assured that he was acute of hearing, and could perceive all noises, which he said hurt him; and had I not tested the fact myself. Having written for him to raise his hand as soon as he heard the sound of a bell, he did so correctly, even when it was rung most gently behind him. It is the deficient attention he pays to sounds which gives one the impression that he is deaf. Then a few minutes later I rang again, but he did not react to the noise by raising his arm a little, until it was very loud. The same want of attention is manifest when one talks to him. He does not make the efforts to understand observed in patients with word-deafness, but remains indifferent; one has to push or shake him in order to make him attend. He hears when one whistles or sings, but does not recognize melodies. When his children sing in his room quartettes, of which he formerly was fond, he tells them to stop, and that they make too much noise. When I played the national anthem before him, he said, "Once more, I shall perhaps recognize it." But he cannot do so.

Speech.—He speaks with absolute accuracy, but with a slight drawl; very rarely, not oftener than would a healthy man, he stops for a word. He finds substantives, even complex ones, and proper names. I showed him a picture, and he said without the least hesitation, "Winterthur." On neither occasion could I detect the least trace of paraphasia.

Writing.—He writes fluently and correctly, and composes long articles for journals, of which I have seen several, and possess one.

Repeating is impossible to him, even when he is told by writing to do so. Thus when I said, "Ich heisse," he fixed his eyes upon my lips and finally brings out an "Ich," but nothing more.

Writing to dictation.—Asked to do so, he said: "But I can't

hear." I nevertheless proceeded to dictate, upon which he remarked: "What a farce to dictate when one does not hear!"

Copying and reading aloud he carries out correctly and fluently.

Intelligence of written language is intact, as shown by what has already been said. He copies an I O U, written by myself, gives it to his wife, remarking, "You see you have got money."

Intelligence perfectly normal. The articles he writes are up to his usual standard and are often published in his newspaper. Advice from his medical attendant informs me that he has remained *in statu quo* up to the present date.

There is in this case only one point which needs discussion here. It is whether the word-deafness produced by the second attack really caused no disturbance of speech. The patient had preserved from his first attack a difficulty with reference to language and writing. If we follow the description of Burckhardt, the second attack brought no change into this condition, and the slight alteration of speech which remained receded completely in the subsequent evolution of the case before I saw the patient. This is in contradiction with the wife's account, who, in answer to my questions, said that the speech had been temporarily worse after the second attack, when writing and reading disappeared for awhile. I have already said that these statements were not trustworthy, having been made three years after the event, whilst the observations of Burckhardt were noted at the time; but I deemed it necessary to mention them precisely, because they seemed to militate against my view. However, even if they were correct, I would adhere to my interpretation, and assume that, in the second attack, the centre A of the diagram had been transitorily affected also. Thus Wernicke's type of aphasia, which includes, besides word-deafness, paraphasia, agraphia and alexia, might have come to be observed for a short time. This state of things would have passed away quickly, and word-deafness only persisted, complicated with a gradually receding trace of the disturbance of speech left after the first attack. This interpretation is obviously more probable than the assumption, that the disturbance of speech was a direct symptom of the focal lesion of the second attack, and was transitory only, whilst the verbal deafness remained. The

opposite course is indeed the usual one, verbal deafness disappearing very much more quickly than paraphasia.

In other respects also the case offers peculiarities. I have already adverted to the possibility of looking upon the patient as slightly deaf. The other cases of word-deafness I have seen were different in this respect; one was apt in them to overlook the symptom, the patients answering all questions, but not appositely, and the danger being then to diagnose confusion of ideas. Our patient, on the other hand, paid no attention to the questions, never returned any answer, and thus gave one the impression of being deaf.

Finally, the persistence of this symptom differentiates the present case from the others. Yet these peculiarities can lend only a high degree of probability to this differentiation; a single case does not warrant certainty, and further instances are required before we can decide. The case seems to be an isolated one; I have suspected from the description of others that they belonged to the same category, but the want of accuracy in the observations has not allowed me to reach a definite conclusion.

I think I have so far shown, that each of the seven forms of aphasia postulated by the diagram are found to exist; but there remains the question, as to whether all cases hitherto observed are reducible to these forms. If we examine a large number of the cases on record, it will be found that the majority present deficiencies of observation which allow the test to be applied to them only up to a certain point. In a large number of instances the probability is that they do really belong to one of the forms. But one readily obtains examples in which this is not the case; they seem to differ in one point or another from these morbid types. Do they constitute a serious objection to my theory? I do not think so; most of them can be shown to be reducible to the schema. I must first advert to the fact, that the seven forms hitherto discussed have their origin in *simple* interruptions; whilst there is no doubt that more than one of the paths may be affected simultaneously. A decisive proof of the reality of such combination types is to be found in "total aphasia," in which

there is complete incapacity to speak (the "logoplegia" of the French), with word-deafness. Here there must be a break in the centripetal as well as the centrifugal portion of the arc. We possess several observations to this effect, in which the autopsies have confirmed this assumption. Under the definition of total aphasia just given, this type will include all cases where there is loss both of speech and of understanding of speech; a combination which will arise not only when M and A are injured, but also when other combined breaks in the two branches of the arc do occur: it will be evident from the diagram that six such combinations are possible, each giving rise to the symptom. It is very doubtful, however, if these theoretical possibilities are all embodied in actual cases; their respective degree of probability can be arrived at only when we know more clearly the anatomical disposition of the nervous paths. We must assume, however, that some other combined lesions occur besides that of A and M; certain observations point to this conclusion. Thus, for instance, the celebrated case of Lordat, which, though I know only through the fragmentary account of Kussmaul, may be explained on the assumption of a simultaneous break in M B and A B. It appears that, like our patient Schwarz, he could read spelling-wise, though without understanding what he did read. Aphasia due to lesion in M B and B A, would be differentiated from that due to lesion of M and A by the preservation of the faculty of reading aloud, of repeating words, and of writing to dictation; but there is no mention made of these functions in the account just referred to.

[Whilst the present article was going through the press, I had the occasion to read the original account of the case of Lordat given by himself.[1] What I have just said has been confirmed, so far that Lordat could repeat words; but no mention is made of any trouble of writing. Certain statements, however, make it doubtful as to whether he was suffering from aphasia, and not from paraphasia only. He says: "There was not only amnesia, but also what I would call paramnesia, that is, an erroneous use of known and

[1] Grasset, "Contribution clinique à l'étude des aphasies."—'Montpellier Médical,' Janvier 1884.

remembered sounds. Thus when I wanted a book, I asked for a pocket-handkerchief. True that immediately after having pronounced this word, I withdrew it; I felt that another was wanted. Another mode of paramnesia consisted in interverting the letters of the syllables in a compound word, of which I had recovered possession. Thus for 'raisin' I said 'sairin'; for 'musulman,' I felt inclined to say 'sumulman.'"

These data are somewhat opposed to those which precede them, and which leave no doubt that, with the exception of a few words, Lordat was quite speechless. I am therefore disposed to assume, that this paraphasia appeared only later, in the period of regression. The explanation given above would thus be perfectly justified; for I have already shown, that paraphasia and amnesia are observed during the healing stage of aphasic lesions in B M.

This supposition is the more plausible, that the quotation from Lordat shows that the word-deafness had already left him; he was conscious of the mistakes he made in speaking.

If the case of Lordat had been from the very beginning one of paraphasia and not of aphasia, it would agree in the main with that of our patient Schwarz (Case I.); and we should explain it simply on the assumption of a break in the path A B.]

Besides such combinations, we may conceive others due to the simultaneous implication of paths on the same side of the arc. The types thus obtained would coincide, to a great extent, with those arising from single breaks; the one due to interruptions in M A and A *a*, however, would present a peculiar clinical picture, distinguished from Wernicke's sensorial aphasia, inasmuch as the understanding of writing would be preserved. I have found no case illustrating this form. Wernicke, however, in his explanation of his type of aphasia, expressly specifies that the faculty of reading may be intact, but the reasons he gives for this fact are quite different from mine.

Further examples of combination may arise from the implication of the nerve-paths interested in the production of written language only, A *o*, A E, and O E (Fig. 4). It is probable that the path O E, at least, is sometimes involved,

for there are cases in which it is expressly stated that the power of copying was affected, as for instance in that of M. Valentin.[1] To judge from the symptoms, this must have been a case of total aphasia caused by a lesion of M and A, with a break in O E.

It is not necessary for me to go through all the possible combinations; many of them are very unlikely to occur in reality.

A second, and very important circumstance which must be kept in view when we deal with cases which appear to militate against our views, is that the several components of the various types disappear at very different rates. A large number of "aphasics" fall under our notice at a variable period after the attack has taken place, when some of the symptoms have already begun to disappear. It is easy to show, by examples, how even a few days may be enough to efface some of the characteristic symptoms of the type; this is what happened, for instance, in our third Case.

We must always bear this fact in mind, when in presence of complicated morbid types of unrecent date. Our task is obviously made more difficult, though we must not despair of elucidating the problem; for there is no doubt that there is a law governing the order of retrogression of the various symptoms, at least when they are not obscured by the interference of indirect focal symptoms. These laws are to be derived from a sufficient number of exact observations. To our small stock of knowledge on this point belongs the fact of the usually rapid disappearance of word-deafness, and the greater persistence of the troubles connected with written language. This fact appears to me to throw light upon a not unfrequent type which does not readily fall in with our classification; I mean that of motor aphasia in which alexia[2] co-exists.

A break in the motor tract can never in itself produce a disturbance in the intelligence of writing; and according to the schema there must then be a lesion of the path A O also. Such a combination, exclusive of the simultaneous implication of a A B, is obviously not impossible, but, as we shall presently

[1] 'Revue Médicale de l'Est,' xii. 6. Quoted by Skwortzoff, p. 63.

[2] By "Alexia" is meant here, not only the impossibility of reading aloud, but also the deficient understanding of written signs.

see, all our actual notions concerning the localisation of aphasic phenomena compel us to admit that it must be a purely accidental one. Now the type I am referring to is so common, according to my own experience, and as shown in Trousseau's *Clinique Médicale*, that we can scarcely admit of an accidental occurrence. An explanation must therefore be found for what would otherwise be a serious objection to the theory proposed in this article.

These cases appear to me to have been instances of total aphasia in which the symptom of word-deafness has already been recovered from; I have actually observed this course of events in a case where the initial total aphasia evolved into the type we are now considering. I have already referred to this case, of which I unhappily do not possess the notes. In other instances a careful investigation has led me to discover remains of word-deafness, consisting in a deficiency manifested by the patient when given more complicated orders to carry out, and corresponding exactly to the similar imperfections noted in the later stages of our Case III. Here is an example of the kind.

CASE V.—*Aphasia appearing during typhoid fever. Doubtful traumatic cause.*

S. T., aged 49, a cabinet-maker, was admitted into the Insel hospital on the 24th of January, 1884. It is almost impossible to hold communications with the patient, owing to the disturbance of his faculty of language. All that could be made out of his previous history, was that he fell ill with typhoid during a great epidemic at Meyringen. One day he fell out of bed upon his head, he remained several weeks (?) unconscious, and was speechless when he came to himself again.

Patient is a fairly strong man; his intelligence appears to be perfectly normal; he tries with a certain degree of success to express himself by gestures. He occupies himself in the wards with the general service and with woodwork. He has made with skill several boxes, and plays dominoes and cards very well. The nurse, however, thinks he is deficient in some ways, because he has failed to carry out certain directions.

Speech.—He is absolutely speechless, with the exception of a few words such as, "yes," "no," "now," "but," "too," "just;" and the sound "gock," which he substitutes on every possible occasion.

Now and then he comes out with a longer exclamation, such as "Herr Jesu Gott," or "Min Gott im Himmel."[1]

Repeating.—He cannot repeat anything said to him except "yes" and "no."

Intelligence of speech.—On a superficial examination the patient appears to understand everything. He points to the right objects when they are named, and carries out simple orders perfectly, shows his tongue, closes his eyes, stands on a chair, shuts the stove, &c. But when the directions are more complicated it becomes evident that he does not understand so well. When asked, for instance, to bring a large bottle standing near a basin, he takes the basin. If simple but unusual things are told him, he does not do them correctly. I asked him one day, on coming near his bed, to show me his nose, and he pulled out his tongue. Suggestive questions are rightly understood by the patient.

Intelligence of writing.—He formerly read and wrote fluently, but now never takes up a book or newspaper, and manifests on careful examination a great loss in his power to understand written language. He apparently manages a few words; for instance, "soldier," "sheep," "horse," "pigeon." He signifies by gestures that he understands the words "wine," "Albert" (the name of his son); short sentences likewise, such as "eyes shut," "mouth open." But beyond this he is incapable of carrying out plain directions written in large characters, though he points to the letters as if to indicate that he knows them. He has great difficulty, however, in picking them out of a box, and seems to have no idea of their alphabetical order. His difficulty in understanding writing is greater than that in recognising spoken words, though the disturbance is obviously analogous in both cases.

Writing.—The patient writes his name correctly, but he cannot write anything else.

To *Dictation* he makes illegible scrawls, except for his own name, which he writes correctly.

Copying.—He copies everything, but in a servile manner, and with all the mistakes made in the model.

Reading aloud absolutely impossible. He knows the numbers, can write them also, even when with two digits. He often makes mistakes in the multiplication table.

In other respects patient is quite well. No abnormality dis-

[1] We tried to determine whether the word-representations could be excited from the higher centre. Objects were shown to him, but he could not give the number of syllables of their names, nor indicate it by pressure of the hand, according to the plan previously described. He did it correctly if the name was given to him, so that he must have understood what he was told to do.

coverable about the head, all functions depending upon cerebral and spinal innervation intact, excepting a weakness of the right hand, which gives 36 on the dynamometer whilst the left gives 55. Patient indicates by signs that this has been the case since his fall. No such difference exists in the lower extremities.

We have to do here with an apparent case of Broca's aphasia in which the behaviour of the patient led one to suppose that he could not read at all; on closer examination a considerable diminution of the intelligence of written language was shown to exist. The understanding of spoken language appeared at first sight undisturbed, but on investigation defects appeared which were obviously remains of a transitory word-deafness; so that the case must be considered as one of total aphasia.

I have observed another case, quite analogous to this, with reference to the troubles in the functions of language; and I believe, as has been already mentioned, that Trousseau's cases must be interpreted in the same way. The inability of the patients to read was here assumed on the evidence of their not taking to books though their intelligence was good. Though they sometimes thought they could read, they persisted in ever using the same volume or the same page; similar phenomena were observed in my two patients. Trousseau, however, does not adduce any more thorough investigation of their intelligence of writing or speech.

If we take into consideration the facts, that the individual disturbances in the functions of language may occur more or less incomplete or partial[1]; and that more or less considerable remains of them may persist and thus complicate the task of elucidating the type to which a case belongs, it will not appear strange that many instances are difficult or impossible to explain. But this forms no insuperable objection to the theory. On the other hand, our hypothesis would be gravely compromised if a whole category of common aphasic disturbances could not find room within its boundaries; and thus

[1] I abstain from entering here into details about such partial disturbances, so as not to complicate a subject already complex enough. Their study must, however, elucidate many points connected with the mechanism of language. A remarkable start in this direction has been made by Broadbent ('Medico-Chir. Trans.' June 1881).

it becomes necessary for us to consider what place the "amnesic aphasia" of authors must occupy.

The first who distinguished between aphasia from loss of memory of words and aphasia from disordered co-ordination of word-movements, or disturbed conduction of volitional impulses, was James Russel.[1] Most English writers, and in Germany Biermer[2] and Kussmaul, have taken this view. For the first form Ogle[3] preserved the name of amnemonic, for the second that of *ataxic* aphasia; but the word *amnesic* has since displaced the former. Our task is to throw light upon the distinctive clinical symptoms by which these two forms of aphasia can be recognised.

Russel gave as a characteristic test that patients with preserved memory of words, but disturbed conduction of volitional impulses, still make notable efforts at speaking. According to this criterion a great number of cases which are now denoted as of amnesic aphasia would not have been considered as such by him, for the patients try hard to speak, and often possess a goodly vocabulary. Nor can I believe that efforts to talk by aphasics who have but a few words left, justify the conclusion that they have the sound-representations of others at their command. Moreover, I think I have shown that in Case V., which belongs to this category, these representations were missing.

Ogle has described as a pathognomonic sign of his amnemonic aphasia that patients can name nothing, or next to nothing, but can usually repeat words spoken before them. On this assumption, the fourth form described by us as depending upon an interruption of the path B M, would rank as amnesic. But it is certain that this definition does not exhaust all that is understood by amnesic aphasia. Most cases of it must receive another explanation.

Let us consider how, from our point of view, an aphasia due to obliteration of the memory of words is distinguished from one due to disturbance in the motor apparatus. As stated above,

[1] 'British Medical Journal,' 1864. Quoted after Spamer.

[2] 'Correspondenzblatt für Schweizerärzte, I. 1871.

[3] "Aphasia and Agraphia."—'St. George's Hospital Reports,' ii. 83-121.—See Virchow's 'Jahresbericht,' 1867, p. 40.

I think that in most motor aphasias—in those, among others, of the true Broca's type—the patient has lost the auditory word-representations, that is to say, cannot awake them voluntarily by an action of their higher centres. The only criterion of the fact is the preservation of the faculty of writing; or else the power of putting together words from letters; or of counting syllables, their number being expressed by signs, or pressure of the hand. These actions are possible to but a small number of the patients, who are described as ataxic aphasics. Kussmaul[1] also considers the power of writing as a sign of the possession of auditory word-images; but as most ataxic aphasics have lost it, he assumed in their case the presence of an ataxic agraphia, that is, of a lesion of the motor-writing tract. I have already shown why I cannot adopt this view; nor can I accept the criterion by which the same author pretends to recognise the faculty of exciting sound-representations in themselves by those ataxic aphasics who have lost the faculty of written language. He says: "The best, but not the only proof of the existence of their faculty is given us by written language. We must assume its existence where aphasics who have never learnt to write, or have lost the faculty of writing, use expressive signs to manifest their thoughts and to answer our questions correctly. Fallacies are obviously dangerous here, and accurate investigations must repeatedly be made in order to ascertain whether the patient actually understands. No signs especially must be made to accompany what we say."

I believe that we must distinguish sharply between the power of resuscitating sound-representations of words voluntarily (i.e. from the concept centres), and the faculty of doing so acoustically (i.e. from the ear), and putting them in communication with the concept-centres. The former involves efferent, or motor, actions; the latter afferent, or sensorial, effects; involving the paths of the diagram B M A and *a* A B respectively. The test of Kussmaul has reference only to the understanding of words (i.e. to the state of the path *a* A B); and which may be lost without destruction of the auditory word-representations, as is shown by Case IV., where we find absolute word-deafness with no disturbance of writing. I have also

[1] Loc. cit. pp. 157 and 158.

found, that though a patient understood speech perfectly, he might have lost every power of voluntarily bringing back the word-sounds (Case II.). In most ataxic aphasias, therefore, I consider that there is, besides the impossibility of expressing any words, a loss of word-memory, that is of the power of sound-revivifications.[1] If we turn to Ogle's amnemonic aphasia, where the faculty of repeating is possible, we find that the power of speech proper is gone.

Loss of voluntary innervation of the auditory word-centre, but not of the motor word-centre, would occur in cases of lesion of the centre A itself, or of the commissure A M. The corresponding types (sensorial and commissural aphasias of Wernicke) described under 2 and 3, do manifest the disturbance of language which has been called amnesia or amnestic aphasia. This trouble we have, like Wernicke, associated with a lesion of the auditory word-representations; and, as the reader will remember, we find in recent cases that the vocabulary is rich (as one would expect when the path B M *m* is intact), but that speech is incorrect, the words used being mutilated or inverted. This symptom we name, after Kussmaul, paraphasia; when it is disappearing, or is but slightly developed, there occur evidences of amnesia, that is more easy to demonstrate when the patient is made to name objects than when he is engaged in ordinary talk; names which occur without effort in fluent speaking, arrest him when he has to find them for objects or persons shown to him. If the path M A be interrupted whilst A remains intact, the auditory word-representations may be called up through the auditory, as well as through the optic nerve; and it is enough to say or show the word to restore it, either for an instant or for a longer period.

These phenomena may be brought into harmony with our schematic representation, by assuming, that for fluent talking the innervation of the auditory word-centre is not necessary, the latter being connected to the path B M *m* only.

The process of finding the name of persons and objects is different. It is easy to convince ourselves by self-observation

[1] Ogle admits that in ataxic aphasia, the memory for words may fail, as well as the power to express them.

that we first seek the auditory representation of the word, from which starts the excitation for the mechanism of articulation; we are not conscious of such a process in fluent speaking. Experience further shows that the same word-amnesia occurs in the type which we have seen to depend upon a break in the path A B (see Case III.). In explanation we must assume that the nervous current, starting from the auditory representation in A, must pass through B to reach M.

The reader may find this very improbable and too complicated. But it would appear as if, in naming objects, the auditory representations once found had to react in consciousness. This variety of language is a much more "conscious" one than fluent speaking, in which we are aware of the sense of what we are saying, rather than of every word we say. Under certain circumstances conversational language is carried on in a similar way to naming, as, for instance, when we use an idiom not quite familiar to us. Here we must seek the words by the complicated process just mentioned; the direct communication between concept and motor centre without co-operation of sound-representation does not exist; the sub-conscious act of speaking is not yet possible. A greater psychical exertion is obviously required, and consequently more fatigue is entailed.

It seems to me questionable, however, to place amnesia on a par with the other phenomena of aphasic disturbance. At any rate, it is a pretty constant accompaniment of the types above mentioned, and of the regressive forms of motor aphasia which pass through a stage where amnesia is observed, as has been shown in the second of the cases we have adduced and in other analogous ones. But it is not a sign of a focal lesion, and appears in more diffuse morbid processes, or where the cerebral circulation is deficient, or as an early sign of the natural involution of the brain.[1] Its occurrence may be explained on

[1] The various forms of amnesia, the senile as well as those observed in aphasic disturbances, do not offer essential differences; they always involve proper names chiefly, then substantives; very rarely verbs, adjectives and pronouns. Kussmaul explains this fact thus: "The more concrete the idea, the more readily the word which expresses it escapes the memory when it is failing. The reason of it is that the representations of persons and things are more loosely connected with their names than the abstractions of their conditions, relations

the assumption, that the resistance offered by the complicated nervous path can be overcome only when the latter is in a normal condition, and when the functions of the cerebrum are fully carried out. Thus amnesia may be found as a residuum after lesions in B M A B, and appear as a symptom of diminished cerebral activity. At any rate, it cannot be accepted as a basis for the classification of aphasias, and the word "amnesic" aphasia had better be abandoned. The expression "ataxic aphasia" is equally unsatisfactory; we have not to do with disturbed co-ordination of word-movements.[1] The misnomer has probably originated in the fact, that movements other than those of speech can be performed by the patient with his vocal organs; but from our point of view, aphasic speechlessness is more of a *paralytic* nature.

We have thus reached the subject of nomenclature, and should advert to the best way of designating the seven simple forms of aphasia we have described. The best plan is to adhere, as far as possible, to the names already accepted and in use. In a preliminary notice I gave last June, I followed the division of Wernicke, and designated aphasia from breaks in the efferent path B M *m* as *motorial*, and that from break in the afferent B A *a* as *sensorial*.

Further consideration led me to a different result. As long as one thought, with Wernicke, that disturbances in the understanding of language were always associated with troubles of speech, the former would be called "aphasias"; but this is not the case when we deal with morbid types in which speech is perfectly intact, such as that described under No. 7. On the other hand, it is obviously necessary to bring together lesions

---

and properties. We represent persons and things to ourselves as easily without as with their names; the sensory representation (Sinnenbild) is more important here than the idea (Sinnbild), the name contributing but little to the knowledge of the person or of the object. Abstract ideas, on the other hand, necessitate the aid of words, which form their sole embodiment. Hence verbs, adjectives, pronouns, and still more adverbs, prepositions and conjunctions, are far more intimately associated with thought. We may imagine that there is a much greater number and combination of excitations of the cerebral cells required for the genesis of an abstract than of a concrete idea; and that there are in consequence far more numerous organic connections between the former and their corresponding word-forming process, than in the case of the latter."

[1] As Sander has already shown. 'Archiv für Psychiatrie,' II. p. 40.

in B A *a* under one appellation. Hence it seems to me more correct to accept the nomenclature of Kussmaul, and group together those affections, designating them as *word-* or *speech-deafness.* The imperfection in the understanding of language is, of course, but a partial symptom in this type; but the same is the case for the speechlessness, or aphasia, symptomatic of a break in B M *m*, and word-deafness bears to the symptoms of a lesion in B A *a*, the same relations as aphasia to those of a lesion in B M *m*. It constitutes the dominating and necessary sign thereof, around which the others are grouped, just as agraphia and the other disturbances centre round the aphasia, in lesions of B M *m*.

We shall therefore do well to reserve the name *aphasia* for the latter class; and, as just mentioned, designate the former by the term *speech-deafness*, or *logokophosis.* Each of these groups fall into three subdivisions: lesions of the centres (M and A in the diagram), might best be said to give rise to *central* aphasia or word-deafness; whilst the others would be distinguished as (*inner* and *outer*) *commissural* aphasia or word-deafness. The disturbance arising from a lesion in M A could provisionally receive the name of *commissural paraphasia,* until its localisation be fixed with certainty in the Island of Reil, when it would be called *insular aphasia.*

I am well aware of the objections which may be raised against this nomenclature. It would have been more rational not to give the name of a prominent symptom to the leading morbid types, and to find new names for them; but I shrank from an attempt involving the substitution of new names for old and familiar ones, and feared only to create more confusion thereby.

Some readers will object, that the nomenclature rests upon a still hypothetical basis; but, in my opinion, it is of assistance to make it rest upon the assumptions involved in the explanation of the problem. Should this explanation, perchance, prove to be erroneous, no harm will have resulted thereby, and we shall have only to alter the names.

We cannot conclude without having previously considered how far we can proceed with the cerebral *localisations* of the various forms of aphasia. We must obviously start from those

data which may be considered as most certain and best defined. The origin of Broca's aphasia in disease of the inferior frontal convolution will scarcely meet with any doubt, and Wernicke's explanation of his sensorial aphasia by a lesion of the neighbouring temporal convolution has been confirmed by subsequent experience. We may thus assume that the points M and A of our diagram occupy these parts respectively. The path M A has been referred by Wernicke, on anatomical grounds, to the region of the insula; and it is of common occurrence to see lesions of this spot cause disturbances of speech; but we cannot as yet affirm that the latter corresponds to our type (through lesion of A M), though Case II. gives support to this view. There we see the clinical association of symptoms accounted for by the destruction of the insula; but the lesion extended to the neighbouring frontal and temporal convolutions, and no data could be obtained concerning the severity of the apoplectic seizure. It is, however, probable that the trouble of speech was an immediate effect of the lesion, because it persisted unaltered more than a month, till death supervened, whilst the other symptoms underwent a more or less considerable retrogression. A case[1] of Meynert's, though somewhat fragmentary, might be adduced here also.

We shall further have to deal with the course of the inner and outer commissures of M and A. Though in the diagram B is represented as a sort of centre for the elaboration of concepts, this has been done for simplicity's sake; with most writers, I do not consider the function to be localised in one spot of the brain, but rather to result from the combined action of the whole sensorial sphere. Hence the point B should be distributed over many spots; and the commissures M B and A B, would not form two distinct and separate paths, but consist of converging radiations from various parts of the cortex to the points A and M (see Diagram 7, p. 478). This admission does not do away with the possibility of interruptions in the commissures B M, B^1 M, B^2 M, &c.; but leads us to expect, that any simultaneous break in them must occur close to their entrance into the lower centres M and A.

Hence we shall have to look for the lesion which produces

[1] 'Wiener medicinische Jahrbücher,' 1876.

inner-commissural aphasia in the white matter of the hemispheres, namely at the base of the third frontal convolution. The anatomical localisation of inner-commissural word-deafness will likewise have to be sought near the first temporal convolution. We have already had to observe more than once,

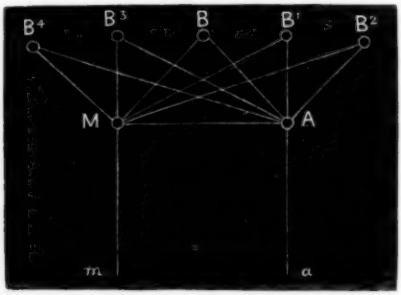


FIG. 7.

that in most published cases, the description of the symptoms is deficient; their morbid anatomy likewise leaves room for many doubts.[1] But though we are still in want of demonstrative evidence, we occasionally find indications in support of our views. Thus in a case of Farge,[2] we find that the patient had right hemiplegia after a severe apoplectic seizure. He could say only a very few words; but could repeat correctly what was said, at least the first words of a sentence. He died, as will be seen in a reference by Nothnagel,[3] twenty days after the fit. The autopsy revealed integrity of Broca's convolution; but in the white matter beneath was found a patch of softening, the size of a small egg.

There is no other case, besides my own, of inner-commissural word-deafness published in sufficient detail to allow

[1] In Hammond's 'Diseases of the Nervous System,' there is a clear case of aphasia, without loss of power to repeat words, quickly followed by death. The case was one of injury, and then were found (1) an ecchymosis the size of ½ dollar in the right frontal lobe; (2) a tear of the right middle meningeal artery, with large hæmatoma. It is obvious that such an observation cannot be utilised for our purposes.

[2] 'Gazette hebdomadaire, 1865,' 44. See Ku smaul, p. 99.

[3] 'Topische Diagnostik,' p. 358.

of a certain diagnosis. The one by Broadbent[1] furnished an autopsy, but as it came under observation several months after the attack, and as a portion of the symptoms had certainly retrograded by that time, there is some difficulty in identifying the type. There were slight word-deafness and paraphasia present; understanding of writing was highly deficient. The patient could write to dictation, copy and repeat words; he died a month afterwards from another attack, when an extensive hæmorrhage took place in the left hemisphere and broke into the lateral ventricle. This fresh lesion made it difficult to recognise the older one; but it appears that the left temporal convolution bordering the Sylvian fissure was intact. A small hæmorrhage was found in the lowest temporal convolution. The focus, the appearance of which made Broadbent connect it with the symptoms, was in the white matter of the temporal lobe, between the posterior end of the Sylvian fissure and the lateral ventricle near the root of the cornu ammonis. The case proves nothing positively, and all which may be said is that it rather harmonises with our hypothesis.

The difficulties are still greater when we turn to the localisation of the paths M *m* and A *a*. We know the terminal points of the former; the third frontal (Broca's) convolution, and the motor nuclei of the medulla oblongata, which enter into activity during the act of speaking. But we know practically nothing of the actual course of this commissure, whether it is bilateral, or runs exclusively between the left hemisphere and the basal organs. The usual view is that the bilateral movements of the lips, tongue, palate and larynx, which enter into play during speech, may be excited from each hemisphere; but that the connection between this process of innervation, and the intelligent concepts and auditory word-representations necessary to the combination of those movements into articulate speech, occurs on the left side only. It thus comes to pass that only the left third frontal and its efferent fibres are engaged in the movements of speech, whilst the corresponding organs on the right side govern the movements of the organs of speech, but have no share in co-ordinating them into the expression of

[1] "Cerebral Mechanism of Thought and Speech," in 'Med. Chir. Trans.;' quoted after Skwortzoff, p. 88. Virchow's 'Jahresbericht,' 1872, II. 45.

words. The consequence is that a lesion on the left side gives rise to a disturbance of speech without apparent loss of motility, whilst a bilateral lesion is required to produce a paralysis of the motor organs.

Now if the fibres from Broca's centre reached the basal organs down the left side only, the usual persistent aphasic symptoms would arise from lesions of the left peduncle or internal capsule as uniformly as they do from those of the centre itself. But we know that this is only exceptionally the case; hence there must be a partial decussation of the speech-tract from left to right hemisphere within the brain itself, so that the left internal capsule does not contain the whole bundle of these fibres. Diagram 8 is intended to represent this fact, and

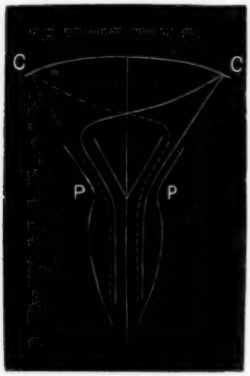


FIG. 8.

shows why the usual lesions of the left internal capsule are not followed with permanent troubles of speech. We see that both hemispheres partake in the function of language,[1] and that bilateral disease is necessary to set up disturbance of speech, combined with deficient movement of the tongue, lips, &c.; such cases, however, are no longer looked upon as instances of

[1] This function is more frequently interfered with by lesions of the deeper portions of the *left* hemisphere, a fact which may be explained on the assumption, that the direct fasciculus is more considerable than the crossed one. The great individual differences observed with reference to this point may rest on corresponding divergences in the relative distribution of fibres in these fasciculi.

aphasia, but have been classified under the name of "Pseudo-bulbar paralysis."

I do not think Wernicke justified when in his last paper[1] he includes under aphasia the speechlessness occurring in the terminal period of progressive bulbar paralysis through total destruction of the motor nuclei. The term aphasia should, I think, be strictly limited to those cases of speechlessness which are unaccompanied with any disturbance of the mechanism of articulation, though I am aware of the difficulty of always accurately defining aphasia from anarthria. When an aphasic is deprived by a fresh right-sided hæmorrhage of the movements of the tongue, lips, &c., we are enabled to recognise the aphasia among the symptoms of pseudo-bulbar paralysis only by our knowledge of how the affection was developed. But occasional difficulties of this sort cannot make me abandon the secure ground afforded by the discrimination between aphasic and anarthric phenomena.

On the assumption that a portion of the fibres decussate, as just suggested, in the hemispheres themselves, an explanation is afforded of all the phenomena which led Wernicke to assume that the right cortex has a share in the production of speech. I hold that no other fact referring to this function is more solidly established than that of the origin of the speech-tract from the left side only. We are not justified in attacking it, as long as we can reconcile with it the data of experience. Now all the facts adduced by Wernicke harmonise perfectly with my view; but whilst his explanation does not account for the regular occurrence of aphasia in disease of Broca's convolution and its non-occurrence in lesions of the left peduncle and internal capsule, these phenomena are the natural consequences of my hypothesis.

Lesions of the deeper portions of the brain, followed by disturbance of speech, moreover, do not bring about aphasia with its characteristics as a "verbal" trouble; the formation of sounds is more or less prominently interfered with. This verbal characteristic of the cortical symptom is a natural

[1] "Ueber die motorische Sprachbahn und das Verhältniss der Aphasie zur Anarthrie."—'Fortschritte der Medicin,' 1884.

consequence of the functions now usually attributed to the cerebral centres in general, and of the innervation of those concerned in language especially. If we assume that Broca's sphere is the seat of acquired motor word-representations (kinæsthetic centre), and that its lesions obliterate these memories, and so give rise to aphasia, it follows that its destruction must affect *words*, and not *sounds*; for those representations acquired during childhood are associated with words only. It is at a much later period of development that we have learnt to decompose words into sounds. As the elements for the formation of words are deposited in the cortical centres of speech, so the dispositions for the production of sounds are made in the bulbar nuclei. The paths, therefore, between Broca's centre and the nuclei will have to undergo a corresponding conversion. The question is now where this conversion takes place; whether low down in the pons, as Wernicke supposes; or, as I think, much higher up, basing my opinion on the fact that disturbances of speech, arising from lesions of the deeper cerebral regions, do not, as just mentioned, partake of a purely "verbal" character.

All these considerations compel us to assume, that only a short extent of the efferent tract from Broca's centre is so constructed as to give rise, on being injured, to real aphasic disturbances. We shall therefore have to look also for the lesion of aphasia without agraphia in the white matter of the hemispheres, though we can as yet scarcely conjecture its localisation. Two new contributions,[1] the results of which, however, are scarcely concordant, point not to a simple radiation of the fibres from the Broca's convolution towards the internal capsule, but to a more complicated arrangement.

The course of the outer commissure A *a* is still more problematical, though one may, in a certain measure, circumscribe the region which it must occupy.

Both ears are capable of receiving and transmitting excitations of speech, but intelligence of language is bound up with

[1] Wernicke, 'Fortschritte der Medicin,' 1884. No. 1. Bitot, "Du siège et de la direction des irradiations capsulaires chargées de transmettre la parole." —'Archives de Neurologie,' 1884, 22, &c.

the activity of the left hemisphere only; so both acoustic nerves must enter into relation with the latter.[1]

The affection due to a lesion in A *a*, characterised by word-deafness, can obviously come into existence only if the irradiations of both acoustic nerves in the left temporal region are broken through.

With reference to the point where the auditory tracts combine to form the path A *a*, we might place it low down in the bulb or cerebellum, and assume that the fibres ascend already, distributed as they are in the cerebrum itself. Such a view would be opposed to the clinical evidence we possess; there are numerous cases of hemianæsthesia through lesion of the posterior part of the internal capsule, in which a diminution of hearing of the affected side existed. Hence the union of the two tracts does not seem to be effected below this point. A unique observation by Vetter[2] of complete deafness of one ear with hemianæsthesia on the same side, of cerebral origin, lends further support to this view. The union, therefore, must take place in the cerebral white matter, and we shall have to look for the localisation of the path A *a* and its lesions in this region, and probably in the temporal lobe.

It is obvious that lesions of the cerebral white matter may not always give rise to the associations of symptoms we have described. Apart from indirect lesions of the cortical centres, complex interruptions in the commissures M B and M *m* on the one hand, A B and A *a* on the other, will give rise to clinical types corresponding exactly to those of Broca and Wernicke.

I have still to advert to the phenomena of *word-blindness*. As we have already seen, this symptom arises in common with others from breaks in different tracts subservient to the function of language. If we were to give to it the same extension as does Skwortzoff, for instance, we obviously could not speak of its being uniformly localised. This condition might rather be

[1] Since a similar distribution must obtain in the right hemisphere, crossed deafness would not be produced in man by destruction of one auditory sphere (as Munk says is the case for dogs). In fact there is no record of observations showing left deafness to be due to a lesion of the right hemisphere.

[2] "Ueber die sensoriellen Funktionen des Grosshirns;" 'Deutsches Archiv für klin. Medicin,' xxxii. p. 498.

fulfilled in cases of isolated word-blindness. Here we have not to do with a simple obliteration of the visual representations of the written signs, with a lesion of the point O in the diagram. These patients can copy, hence the path O E must be intact; and from what we know of the cortical distribution of the optic tract in man, it is certain that the visual memories of words must be stored up in bilateral spheres. Isolated word-blindness is, as has been said by other observers, the result of a break between the visual and the auditory centre of word-representations; that is to say, of a break in the path O A after the union of the converging tracts from both occipital lobes. The observations bearing on the point are few, but they support this conclusion; the anatomical subtratum of word-blindness has been found to be a focal lesion in the neighbourhood of the left inferior temporal lobe.

Though I have ventured the above remarks concerning the probable localisations of aphasic disturbances, I am well aware of the restricted foundations on which we may safely build, and what a space theoretical reasoning has still to occupy in the discussion. If I have, nevertheless, not kept my views to myself, it was on the principle, that we must not recoil from the consequences deducible from our hypotheses. In proportion as we draw these conclusions, we shall obtain the necessary data whereby to correct, or if need be abandon, them. Again, no simple observer can collect material enough to accomplish this task, which requires the co-operation of many; and under these conditions even erroneous assumptions may prove of advantage in the search for truth.

ON THE NERVOUS LESIONS PRODUCED BY LEAD-POISONING.

(*From the Pathological Laboratory of the Owens College.*)

BY ARTHUR ROBINSON, M.B., C.M.

THOUGH cases of lead-poisoning are of frequent occurrence, up to the year 1880, when Dr. Moritz[1] published his paper on Lead Palsy, only seventeen cases had been placed on record in which a satisfactory post-mortem search for the cause of the paralysis had been made; Dr. Moritz's case brought the number up to eighteen.[2] Since 1880 two other cases have been published. One by Oeller,[3] who found the following changes in the nerves and spinal cord of a man, aged 40, who had worked in lead for a year and three months, and who had suffered from paralysis of the extensor muscles of the wrist and fingers of the interossei of the hand, and of the muscles of the thenar eminence.

Spinal Cord.—Small hæmorrhages were found in the middle of the anterior horns, and at the junction of the anterior and posterior horns, in the anterior longitudinal fissure round the large blood-vessels, and also in the white substance of the cord. In the cervical enlargement, from the fifth to the eighth cervical nerves, small spots of softening were found. Spots of softening were also found in the middle of the anterior horns and in the anterior part of the posterior horns. In these positions the ganglion cells were small, atrophied, highly-pigmented, homogeneous, hyaline, and devoid of processes;

[1] 'Journal of Anatomy and Physiology,' 1880.

[2] Lancereaux, 3; Gombault, 1; Westphal, 1; Tiburtius, 1; Friedländer, 1; Ollivier, 1; Zunker, 1; Eisenlohr, 1; Vulpian, 1; Déjérine, 5; Monakow, 1; Moritz, 1.

[3] Oeller, Thesis, Munich, 1883.

they stained badly, and no nuclei were visible in them. Others of the ganglion cells contained large vacuoles. There was a number of spider cells.

The white substance of the cord was only affected in a subordinate degree. Some of the fibres of the musculo-spiral nerve were atrophied, and there were the usual changes in the muscle. Oeller concludes, that the central changes were the primary and essential part, and that the hæmorrhages, following on the general arterial degeneration, were the cause of the softening.

The second case is recorded by Duplaix.[1] The patient was a man, aged 47. He had been a painter for many years, and had been very feeble for a year. There was no trace of syphilis or alcoholism.

The patient noticed, when the feebleness first affected him, that his limbs rapidly lost flesh, and finally the emaciation extended to his face and trunk. The muscular feebleness increased, and at last he was unable to leave his bed. Sensation was normal. He was affected with purpura, œdema of the lungs set in, and death rapidly followed. The post-mortem showed granular kidneys, a small heart, and atrophied muscles.

The spinal cord and the nerves were carefully examined, marked changes being found in both.

Spinal Cord.—The neuroglia was increased in amount, forming bands of tissue enclosing spaces filled with nerve-tubes.

The nerve-tubes were also separated by fine fibrils of connective tissue and many round nuclei. The vessels throughout the whole cord were sclerosed and surrounded by a dense connective tissue.

The nerve-cells in the anterior and posterior horns were unchanged. In the anterior roots, distinct changes were discovered in the structure of the nerve-fibres. The alteration did not extend over the whole length of a fibre, the affected portion being continuous at either end, with an absolutely healthy portion.

The disease usually extended over three or four internodal

[1] Duplaix, 'Arch. Gén. de Méd. 1883,' vol. 2.

segments. Rarely was one segment affected alone. In fact, the changes were those described by M. Gombault, under the name "phase of restitution."

The myelin sheath was everywhere present, but some portions of the fibres were thinner than others, the diminution in volume affecting both the axis-cylinder and the myelin sheath. Nerve-fibres were also seen exhibiting swelling of the white sheath, and also of the axis-cylinder, and in some places there were empty tubes, the nuclei of which had proliferated. The posterior roots were healthy.

The nerves showed both degeneration and restitution. The muscular fibres had atrophied, and their nuclei had proliferated.

The following case was admitted into the Manchester Royal Infirmary, under the care of Dr. Leech, whom I have to thank for kindly permitting me to make use of it, in December 1882. The patient, J. C., a man, aged 30, had been a painter for fourteen years, but for three years before his admission he had nominally been a cabinet-maker, though he still occasionally did a little painting.

He had his first attack of "drop wrist" in the summer of 1877; it continued through the winter, but he recovered in the following spring. Every winter after his first illness, the attack returned and laid him up until the spring.

He was troubled with frequent micturition, but had never had colic or cramps.

The muscles of both forearms were very much atrophied, the extensors of the wrists and fingers were completely paralysed, while the flexors retained a fair amount of power. The muscles of the thenar and hypothenar eminences were also atrophied, and the patient could not oppose his thumbs. He could pronate, but could only partially supinate both forearms. The peroneus brevis and longus muscles on the left side were atrophied and paralysed, and those of the right side were affected with paresis. All the atrophied muscles showed the reaction of degeneration when tested with electricity.

The patient was treated with iodide of potassium, and the interrupted and continuous currents were applied to the affected muscles. He gradually improved, the atrophied muscles

increased in size, and to a certain extent regained their power. He was dismissed in March, 1883, very much improved.

He was readmitted in June 1884, suffering from œdema of the lower extremities, and from effusion into both pleuræ; his muscular system was almost in the same state that it was in when he left the hospital in 1883. His urine contained albumen and coarsely granular casts. He improved under treatment, and left the hospital in March; but the œdema returned, and he was readmitted at the end of the same month. Ascites developed, and he died on the 14th of August, 1884.

A post-mortem examination was obtained, and the spinal cord, together with some portions of the musculo-spiral nerves, were removed, but, unfortunately, none of the atrophied muscle was kept. I examined the cord carefully, by means of sections taken from the various regions, and stained some with carmine alone, others with osmic acid and carmine.

There was no visible abnormal change in any part of the nervous matter. The nerve-tubes in the white columns were intact; there was no swelling of the axis-cylinder, or degeneration of the myelin.

The nerve-cells in the grey columns did not appear to be reduced in number, or to be at all atrophied. Their processes were well marked, and their nuclei distinct.

There was no marked increase of the neuroglia.

There was well-marked hyaline degeneration of the walls of the arteries, both in the grey and white substance of the cord. The degeneration affected, principally, the middle and outer coats of the vessels. The cellular elements were swollen, their outlines in many places being lost; the nuclei were not visible, and they did not stain with either carmine or logwood.

The musculo-spiral nerve was examined by means of transverse and longitudinal sections, and preparations were also made by teasing out the fibres with needles. The sections and teased-out specimens were stained with carmine and osmic acid.

The axis-cylinders were everywhere distinct; there was no fragmentation of the myelin sheaths, except in a few instances, where the breaking up was evidently due to the method of preparation. There was no proliferation of the nuclei of the

sheaths, nor was there any increase of the surrounding connective tissue. The walls of the small arteries in the nerve-sheaths had undergone a hyaline change similar to that noticed in the arteries of the cord. The radial nerve was not kept.

The kidneys were small, the capsule was adherent, and when separated carried with it portions of the kidney substance.

Microscopically they presented, in a well-marked form, the changes of advanced cirrhosis. The newly formed connective tissue was most abundant and most dense round the interlobular arteries.

When lead-poisoning first attracted observation, attention was chiefly directed to the affection of the extensor muscles, and Gusserow[1] attempted to explain the paralysis by supposing a particular affinity of lead for the muscles; but this hypothesis was completely destroyed when Heubel[2] demonstrated the fact, that the muscles contained less lead than the bones, kidneys, liver, brain or spinal cord; and some researches undertaken by Bernhardt,[3] with a view of discovering the relative amounts of lead contained in the paralysed extensor and the normal supinator muscles, gave negative results.

No one would undertake such investigations now, for it is well known that the action of lead, as a poison, is not local but general; the general effect being evidenced by the various encephalic symptoms due to lead, by optic neuritis, amaurosis, hemianæsthesia, and other anæsthesiæ of irregular distribution; tremors, and paralysis, not limited to one set of muscles, but affecting the muscles of the body generally;[4] colic, cramps, and affections of the kidneys.

Though the paralysis produced by lead is by no means limited to the extensor muscles, still they are generally the first to show definite changes. The researches of Onimus,[5] carried out on the muscles and nerves after death, point to an

[1] Virchow's 'Archiv,' 1881.

[2] 'Path. u. Symptom. d. Chem. Bleilähmungen,' 1871.

[3] 'Archiv für Psychiatrie,' iv., p. 616.

[4] Buzzard, 'Brain,' No. 1. Duplaix, 'Archiv. Gén. de Med.'

[5] "On the Excitability of the Nerves and Muscles after Death" ('Journal de l'Anatomie,' No. 6, 1880).

explanation of this fact. He found that the extensor muscles and nerves lost their excitability before the flexor muscles and nerves, and those of the upper extremity before those of the lower. Again, the fact, first pointed out by Dr. Semon, is now well recognised, that the abductor muscles of the larynx always become paralysed before the adductors, in central or peripheral disease of its nerves. These observations point to the conclusion, that the extensor and abductor muscles and nerves possess a less power of vital resistance than the flexor muscles and nerves, and consequently, under the action of a general poison, they will be the first to succumb.

Concerning the lesion which is the direct cause of the paralysis three views are at present held.

Friedländer[1] holds that the primary change is a myositis, induced by the lead, and that this is followed by a neuritis, which ascends to the cord.

The second hypothesis, supported by Zunker, Leyden and Ferrier, is that the primary change is a peripheral neuritis. Lastly, Remak, Vulpian and Déjérine believe that lead produces a poliomyelitis anterior.

So far the results of the published cases appear to favour the second view. We know that lead acts injuriously on the nerve-cells in the brain, producing in many cases serious symptoms, and, not rarely, death. There is no reason, therefore, why it should not have a direct action on the nerve-cells of the cord. Further, a poison may seriously interfere with the function of a nerve-cell, and yet, at first, produce no visible change in it; alcohol and strychnia act in this manner.[2] But, while the nerve-centres are subjected to the direct influence of the poison, and must also be injuriously affected by the general arterial degeneration, the nerve-fibres are at a still greater disadvantage, for, besides being directly acted upon by the lead, and suffering from the arterial degeneration, they will also be affected by the loss of the trophic influence which, in a state of health, the nerve-cells exert over them.

Thus it appears probable, that as the nerves suffer in a double manner, as compared with their centres in the cord,

[1] Virchow's 'Archiv,' 1878.

[2] Cf. paper by De Watteville, "The pathogeny of lead paralysis," 'Lancet,' 1880.

alterations will proceed more rapidly in them than in their centres, and consequently visible changes will, as a general rule, be first recognised either in the nerve-trunks, or their peripheral terminations. So far the published cases support this view, for in 11 out of the 21 cases, changes were found only in the nerves and muscles.[1] In the case I have reported above, no changes were discovered either in the cord or the trunk of the musculo-spiral, though it is very probable that some degeneration would have been found in the radial nerve or its peripheral terminations, had they been kept.

In 4 cases, degeneration was found both in the nerves and the anterior nerve-roots, the cord not being affected;[2] while in 5 cases the cord, as well as the nerves and their roots, was affected:[3] and in one of these, namely that described by Zunker, the changes in the cord, which consisted of localised spots of softening, did not correspond to the centres, from which the diseased nerves sprang. It is to be noted, further, that, in all the cases in which visible alterations were discovered in the cord, the symptoms had been present for a long period of time.

Finally, I have to thank Dr. Dreschfeld for his kind assistance in the examination of the microscopical specimens.

[1] Lancereaux, 2; Gombault, 1; Westphal, 1; Tiburtius, 1; Ollivier, 1; Eisenlohr, 1; Déjérine, 3; Moritz, 1.

[2] Friedländer, 1; Déjérine, 2; Duplaix, 1,

[3] Lancereaux, 1; Zunker, 1; Vulpian, 1; Monakow 1; Oeller, 1.

Clinical Cases.

CASE IN WHICH ATTACKS OF INTERMITTENT TONIC MUSCULAR SPASMS, IMMEDIATELY FOLLOWED BY COMPLETE TEMPORARY PARALYSIS, HAVE FREQUENTLY AND PERIODICALLY OCCURRED DURING THE ENTIRE LIFE OF THE PATIENT, THE HEALTH IN THE INTERVALS BEING NORMAL.

BY A. HUGHES BENNETT, M.D.,

Physician to the Hospital for Epilepsy and Paralysis, and Assistant Physician to the Westminster Hospital.

Antecedent History.—The patient is a young woman aged 17. Her family history is unimportant.. The mother states that during utero-gestation she herself suffered from nervous symptoms as the result of fright, from being chased by a mad dog. She nursed the child for a year, but it was always very delicate and undersized, and was unable to walk till it was three and a half years old.

At birth the patient is stated to have been apparently healthy, but three days afterwards she was seized with a "fit." After this first attack, which was of a convulsive nature, she had no definite illness, but was subject to frequent "faintings," in which she lay for a short time motionless, and "turned up the whites of her eyes." When three years old, it was for the first time observed that in these seizures the limbs were "drawn up." These periodical attacks have continued since, but have become more frequent and severe. Two or three years ago they were accompanied by screaming and other apparently violent hysterical symptoms, but of late these have disappeared. The catamenia have never occurred. In the interval between the attacks the physical health has been good, but mentally the patient is considered deficient, and although she has been to school, she cannot read or write like other girls of her age.

Present condition—A. *During the intervals of the attacks.*—The patient is pale and delicate-looking, and seems several years younger than her age, but all the organs and functions are normal, the body is well nourished, and the general health is good.

She has a peculiar sardonic expression, owing to the lower lip being tightly contracted round the teeth, over which the upper lip projects. There is marked flattening of the skull in the region of the posterior fontanelle. The movements of the eyeballs are somewhat restless and unsteady, and there is difficulty in fixing them on any object, but there is no strabismus or otherwise deficient action. The pupils are equal and normal, and contract to light and accommodation. Vision is natural. The mouth and face are symmetrical, but their movements are accompanied by slight choreic-like twitchings. The tongue is straight, and its actions are apparently normal, with the exception of a slight restlessness on movement. The articulation is not very distinct, but without marked peculiarity. The patient is cheerful, and willingly assists in the work of the ward; she answers questions of an ordinary kind, and conducts herself in a way which has not caused any special comment from the nurses. Her intelligence is, however, evidently defective. She is slow and stupid, childish in her manner, cannot give an account of her illness, has a bad memory, and cannot read or write. The special senses are all intact. The appearance, movements, and strength of the limbs are normal in every respect, except that there is a constant slight restless or choreic-like movement of all the extremities. The grasp of the hands and all the actions of the upper limbs are as in health. Locomotion is normal, and the patient can walk and move about like other persons. There is no rigidity or wasting of the muscles, which have a natural appearance. Sensibility of the skin is everywhere normal. The knee-jerk phenomena are distinctly increased, but the plantar reflexes are normal. The mechanical irritability of the muscles is not markedly augmented. The electrical reactions are somewhat difficult to determine with delicacy, owing to the nervousness of the patient, and to the choreic-like movements of the limbs. They appear to be everywhere normal, with the following modifications. 1st. Stimulating the *muscles* with the galvanic current causes more vigorous contraction with the same strength of current, than when the trunk of the *nerve* which supplies them is acted upon. 2nd. In both muscles and nerves the AOC is slightly but distinctly greater than the ACC; the KCC being greater than either, and KOC is not obtainable by the strongest currents which could be tolerated.

B. *Condition during the attacks.*—This case having been under observation for upwards of a year, the condition during the so-called attacks may be summarised as follows. The patient, while in her usual healthy state, suddenly, and without apparent cause, experiences a peculiar sensation which she cannot describe, but which indicates that a seizure is imminent. She at once goes to her mother, who observes "the veins in her forehead swelling." A few minutes afterwards a sudden tonic spasm of certain muscles takes place, during which they become tense and hard, and which contract the limbs in different directions, so as to cause a variety of distortions. The parts thus affected are immovable and rigid, and their position cannot be changed by voluntary effort or even by considerable active force. When the legs are thus attacked, the patient falls down, owing to the violently produced deformity which ensues. There is no loss of consciousness or intellectual disturbance, and only when the contractions are unusually severe is there pain of a cramp-like character. This condition having lasted for about five minutes, the rigidity rapidly relaxes, and is succeeded by complete paralysis in the same distribution, in which the limbs hang flaccid, useless, and incapable of any voluntary movement. This state continues for about five minutes, after which gradually the muscles regain their power, and in from ten minutes to a quarter of an hour the patient is in a perfectly normal condition. This constitutes one attack. For the past few years the rule has been for these seizures to occur at irregular intervals, many times a day for about a week, after which there is a fortnight of complete freedom, during which time the patient is quite well. Then occurs another week of attacks, and so on, with fairly regular occurrence. These seizures are not all of equal severity. Most commonly one or more limbs only are affected; but occasionally the entire body is in a state of rigidity, every voluntary muscle apparently being in a state of violent contraction. Frequently a single limb only is involved; usually both extremities of the same side; sometimes one arm and the other leg; more rarely the two arms, or the two legs; and occasionally the entire body. When the limbs are the subject of this spasm the effects are not always the same, the muscles do not seem to be always attacked with the same proportionate severity, and consequently the deformity varies, and this may occur during different attacks without apparent definite order. Some of the chief attitudes assumed are portrayed in the annexed drawings, which, however, do not give a complete idea of the intense muscular contraction which deforms the limbs. In the upper extremity there are three prominent types. 1. The upper arm adducted and rotated

inwards and backwards; the forearm extended and pronated, the wrist flexed, the proximal phalanges of the fingers flexed and their two terminal phalanges extended; the thumb extended and abducted; the entire limb projecting in a straight line posteriorly, the palm of the hand looking backwards and upwards (Fig. 1). 2. The upper arm abducted

Fig. 1.

and at right angles to the trunk; the forearm flexed; the wrist, fingers and thumb flexed; the clenched fist resting on the shoulder (Fig. 2). 3. The upper arm adducted, with the elbow close to the side; the forearm flexed; the wrist extended; the proximal phalanges of the fingers and thumb extended; their distal phalanges flexed; the hand resting on the shoulder, the palm upwards (Fig. 3).

In the lower limb there are also three chief types of de-

formity. 1. The thigh completely extended; the leg flexed; the foot extended; the toes flexed; the plantar aspect of the foot looking upwards and forwards (Fig. 1.) 2. The thigh partly flexed and adducted, so that the knee crosses the other limb; the leg slightly flexed; the foot inverted; the toes flexed. (Fig. 2.) 3. The thigh, leg, foot and toes extended, so that the limb is in a state of rigid straight extension, the toes

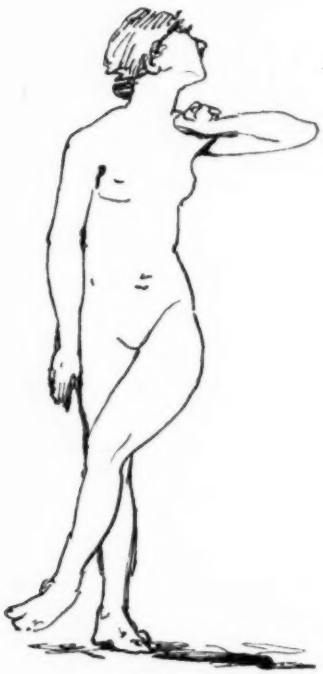


Fig. 2.

Fig. 3.

pointing downwards, the foot being in the position of talipes equinus. (Fig. 3.)

Even these three main types of deformity in the limbs undergo various minor modifications. When the entire body is attacked, one or more of the positions described of the limbs may be assumed, and are generally different in the two sides, so that a most irregular appearance is the result. (Fig. 4.) In addition, the muscles of the face are contracted and rigid, and

the countenance distorted in a variety of ways; the eyelids are sometimes forcibly closed, sometimes widely open. The eyeballs generally are fixed upwards and inwards, so that only the white of the eyes can be seen. The neck is stiff and rigid, the head being generally forced backwards till it touches the back. There is evidently obstruction to the breathing, possibly due, partly to contraction of the muscles of the neck, and partly to fixation of those of the thorax. The spine is arched and in a condition of complete opisthotonus. During such an attack the senses are acute; the muscles are all hard and tense, there is severe cramping pain, and considerable dyspnœa. Swallowing is impossible, speech is prevented, and the skin is bathed in profuse perspiration. This is succeeded by complete temporary general paralysis, in which the limbs hang limp and perfectly useless. The nature of any seizure cannot be predicted, and the extent and distribution of the deformities succeed one

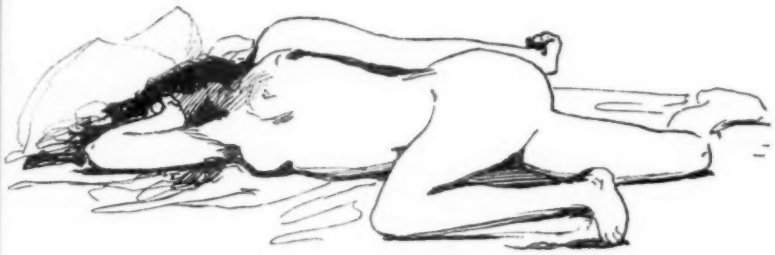


Fig. 4.

another without method or regularity. The precise order in which the individual muscles were attacked could not be determined with any degree of exactitude on a sufficiently extensive scale to form any generalisations, as the physician himself did not frequently observe the entire paroxysm, and the statements of the nurse were not sufficiently accurate. As far, however, as could be ascertained, the most common sequence of events was as follows: Contraction begins in the left hand, spreads upwards to the forearm and arm; then involves the left foot, and subsequently the rest of the lower limb; afterwards the face, eyeballs and head, which last is drawn backwards and towards the left. No exciting cause for the paroxysms could be determined, except that they were liable to be induced by mental emotion and excitement. They never took place during sleep, and were not brought on by voluntary motion or artificial stimulation.

Progress of the Case.—No essential change has taken place

in the last fifteen months, during which the patient has been under observation. The attacks still occur with their former frequency, but of late their severity has somewhat diminished. Treatment of a variety of kinds, including tonics, sedatives, and electricity, has been followed by absolutely negative results. During the spasmodic attack, friction of the affected parts usually gave relief. For the past few months the general health of the patient has been unsatisfactory, and there is a suspicion of incipient phthisis: otherwise her condition is as before.

Commentary.—The clinical picture just portrayed does not exactly correspond with any of those diseases to which a distinguishing name has been given. I have therefore been compelled to describe the case under the appellation of its chief and characteristic symptoms, namely, attacks of intermittent tonic muscular spasm, immediately followed by complete temporary paralysis beginning three days after birth, occurring frequently and with almost regular periodicity throughout the entire life of the patient, the health between the seizures being normal. Although the sequence of events detailed does not in all respects answer to any of the typical descriptions of our text books, it at the same time presents certain features in common with some of them. These it may be well to notice, with the view of determining whether the affection under consideration may not be a modified form of some well-known disorder.

In so-called "Thomsen's disease," for example, there are temporary attacks of cramp which may have existed during the whole life of the patient; but between this and the condition of our patient there is an important distinction to be made. In Thomsen's disease the muscular spasm is directly caused by voluntary efforts, and by them alone. In the case before us the contractions are spontaneous, and in no way affected by active movement.

To the severe forms of Tetany this case bears some resemblance. The intermittent nature of the spasm is common to both. On the other hand, these two affections diverge in many important particulars. The disease under consideration having begun immediately after birth, and continued ever since; the attacks being unaffected by external irritations, and the characteristic hyper-excitability of the nerves on electric stimulation being absent; the temporary but complete paralysis after each seizure, and the impaired mental faculties; all warrant us in removing it from the typical category of Tetany.

To the contractures of hystero-epilepsy this case might at first sight appear to bear a likeness, and the annexed drawings of some of the deformities presented by the patient are some-

what similar to those delineated in the beautifully artistic representations of M. Richer in his great work.[1] Here, however, consciousness was never lost during the attacks, and there was a total absence of that train of other symptoms characteristic of hysteria. It is true that there was a somewhat vague account of the girl's conduct, lasting for a few months only, which suggested that disorder, but this, if present, was probably a complication of her already existing malady.

This case must therefore stand by itself, as it cannot be placed under the head of any of those affections in which there is a typical clinical history, condition, or progress. Although its leading features have already been detailed, I may now summarise the salient points, those which are unusual, and which render it specially worthy of attention. 1. The disease began at the early age of three days, and has continued without intermission for seventeen years. 2. The most prominent symptoms were attacks of intermittent tonic muscular contractions of varying severity, and involving varying groups of muscles. 3. The seizures of muscular contractions were periodic, and occurred at almost perfectly regular intervals during every third week of the patient's life. 4. The muscular contractions occurred spontaneously without apparent cause, and were not induced by voluntary motion, by friction or other external stimuli, and the electrical excitability of the nerves and muscles was not increased. 5. The muscular contractions were, as a rule, painless. 6. The contractions followed no regular order, and were accompanied by no constant or special deformity, such as the so-called gynecological hand. Most frequently the spasm began in the left fingers, extended from them up the arm to the face and head; from thence to the foot, and up the leg and thigh of the same side. 7. The attacks of spasm were immediately followed by complete but temporary paralysis, which was exactly confined to the same regions, and in which there was no trace of muscular rigidity or voluntary movement. 8. The general health of the patient in the intervals of the attacks was robust throughout, except the slight choreic movements on voluntary motion, and some degree of mental deficiency.

The pathology of this condition must remain for the present a matter of speculation. It is improbable that the muscles themselves are diseased. There is no evidence that the nerves are affected. The seat of lesion must therefore be situated in the nervous centres, probably in the brain, and possibly in the motor areas of its cortex. Its nature we cannot demonstrate. It is scarcely likely that any gross organic degeneration is

[1] 'Etudes Cliniques sur l'Hystéro-Épilepsie ou Grande Hystérie.' Paris, 1881.

present. We must therefore assume the disturbance to be of dynamic origin and due to delicate molecular trophic changes, the existence of which is hypothetical. Hence this disorder may be looked upon as a neurosis. The intermissions, the periodicity, and the paralysis following the spasms, would seem to indicate a continued irritation of ganglionic cells in the grey matter, which causes explosions at intervals, as evidenced by the motor paroxysms, which excess of energy is followed by exhaustion in the shape of paresis. The occasional order in which these phenomena take place is suggestive of this irritation being situated in the cortical substance of the cerebrum.

A CASE OF MULTIPLE TUBERCULAR TUMOURS, ONE OF WHICH WAS SITUATED IN THE LEFT CRUS CEREBRI AND CAUSED PARALYSIS OF THE THIRD NERVE OF THAT SIDE, WHILE ANOTHER WAS SITUATED IN THE SPINAL MEMBRANES ON THE LEFT SIDE, ON A LEVEL WITH THE JUNCTION OF THE CERVICAL DORSAL REGIONS, AND PRODUCED PARALYSIS OF THE MOTOR AND SENSORY BRANCHES DERIVED FROM THE SEVENTH AND EIGHTH CERVICAL AND FIRST DORSAL NERVE-ROOTS.

BY JAMES ROSS, M.D., LL.D., F.R.C.P.,

Senior Assistant-Physician to the Manchester Royal Infirmary.

FOR the notes of the following case I am indebted to my clinical clerk, Mr. O. Kauffman.

C. P., a widow aged 24 years, was admitted on December 29th, 1883, into the Manchester Royal Infirmary, under the care of Dr. Ross.

The patient states, that when an infant of about two years of age her right arm was injured by a fall. From this accident dated a protracted illness, from which she did not recover until pieces of bone were removed by operation from her right forearm. About the same time abscesses formed in the neighbourhood of the right knee, and these had to be opened. From that time forwards she enjoyed pretty good health, until four or five years ago, when her left ankle became diseased, and three years ago Mr. Lund had to amputate the leg below the knee. Soon after recovering from the operation the patient married, and had a child eleven months ago, which is now living and healthy.

A few weeks after the birth of her child the patient began to suffer from a constant, deep-seated, throbbing occipital headache. During the following six months headache was the only symptom of which the patient complained, but at the end of that time she found on awaking in the morning that the left eyelid had "fallen down" and that she was unable to open that eye.

Soon afterwards her left hand became suddenly paralysed,

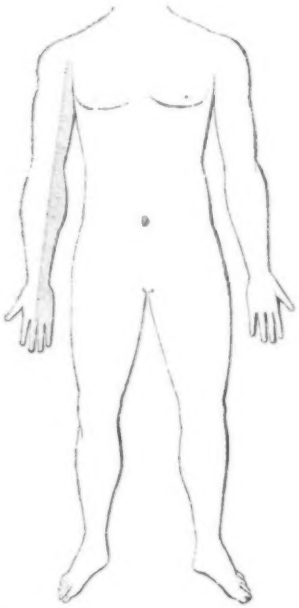
whilst at the same time she felt a numbness of the inner aspect of the forearm, and the inner half of the hand, and of the little and ring-fingers.

Present condition.—The patient is a small and pale woman, with a pimply and blotched face. The back of the right elbow-joint and of the forearm is covered with large cicatrices, and the joint is partially ankylosed. The right knee-joint is also covered with cicatrices and ankylosed, and the patient is at present suffering from strumous disease of the great toe of the right foot. The left leg presents a healthy stump after amputation about the middle of the calf.

There is complete ptosis of the left eyelid, and on raising the lid by the finger, the eye is seen to be directed outwards, while the power of directing it inwards, upwards, and downwards, is almost completely lost. The pupil is in a moderate condition of dilatation. There is no ptosis of the right eyelid, but the movements of the eyeball appear to be somewhat restricted, but it is difficult to know whether this is due to an actual paralysis or to the peevish disposition of the patient, who can hardly be induced to do anything she is told. The grasp of the right hand is comparatively good, and there is no evidence that it is the subject of any paralysis. The muscles of the right lower extremity are wasted, and all the movements are feeble, but there is no evidence of special paralysis. The condition of the tendon-reactions could not be ascertained on the right side of the body, owing to the cicatrices over the elbow and knee and the ankylosis of those joints. The grasp of the left hand, the side on which the ptosis exists, is completely lost. When the patient is asked to grasp any object, the hand is feebly hyper-extended at the wrist, but the fingers do not become in the slightest degree flexed. The muscles of the thenar and hypothenar eminences, the interossei and lumbricales, the long flexors of the fingers and the flexors of the wrist are much atrophied; and even the extensors of the fingers, thumb, and wrist are wasted; so that of the muscles of the forearm and hand the supinator longus alone has retained its plumpness and power, and this is the only one which contracts when its tendon is struck. The hand presents the well-known claw form produced by atrophy and paralysis of all its muscles. The muscles of the right thigh are considerably wasted, the patellar tendon reaction cannot be elicited, and the movements of the limb are feebly performed, but it is doubtful whether this is due to general weakness or to a special paralysis. An objective examination shows that there is almost complete anæsthesia of the skin over the back and front of the inner half of the hand and two and a half fingers, and of the inner aspect of the forearm, while there is

partial anæsthesia of the inner aspect of the upper arm. (Fig. 1.) There is no evidence of anæsthesia of any other part of the body, but the patient complains of pains in the lower extremities. The fundus of each eye is found to be normal on ophthalmoscopic examination, and there is no disorder of any of the special senses. The urine is acid, specific gravity 1017, and free from albumen. There is no evidence of disease of any of the internal organs.

Fig. 1.



The shaded portion shows the distribution of Anæsthesia in the Case of C. P., except that it is inadvertently represented on the right, instead of the left side.

Jan. 8th.—A few days after admission the urine began to dribble, and it is now passed in bed; but the fæces are still retained. The patient complains of some difficulty in swallowing, and states that she has to take a drink after every morsel of solid food swallowed. Food collects in both cheeks, so that she is sometimes obliged to remove it from between the cheeks and the teeth with the aid of the finger. Slight

difficulty is experienced in the articulation of words. The nerves and muscles of the left arm react to a faradic current of moderate strength; but the extensors and flexors of the forearm, and the intrinsic muscles of the hand, contract to a weaker galvanic current on anodal than on cathodal closure, these muscles thus manifesting Erb's partial reaction of degeneration.

Feb. 9th, 1884.—Since the date of last report the patient has suffered from headache and vomiting. She is restless and wakeful at night, and shouts out so continually that she has become a nuisance in the ward. During the day she is drowsy, and can hardly be got to reply to any question. The few words which she utters are so indistinct as to be hardly intelligible. The patient was now discharged.

March 8th.—C. P. was re-admitted to-day. She is now cold and almost pulseless, and passes her urine and fæces in bed. She only mutters a few words, and these are so indistinctly articulated as to be almost unintelligible. The muscles of the right hand are now slightly atrophied, but it is impossible to subject the patient to an electrical examination. She gradually sank, and died on the night of her second admission.

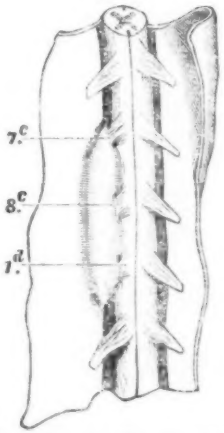
Autopsy.—The post-mortem examination was conducted by Dr. Maguire on March 10, about 36 hours after death, and the following is his report: "The body is much emaciated. Rigor mortis present—post-mortem-staining on back. Pupils equal and of ordinary size. Orifices of body normal. Left hand is claw-form, and shows atrophy up the thenar and hypothenar eminences. Right arm shows old cicatrices about elbow and ankylosis of the joint. The hand is also claw-shaped, and there is atrophy of the thenar and hypothenar eminences, but less marked than on the left hand. Left leg amputated below the knee, and wound completely healed. Right knee ankylosed, and shows old cicatrices about the joint. Subcutaneous fat scanty. Both pleuræ show old adhesions. The left lung shows some patches of pneumonia in stage of hepatisation near the root. The right lung is congested, but otherwise normal. No tubercles to be seen in any part of the lungs. Pericardium normal. The right heart contains post-mortem clots, the left heart is empty. Valves normal.

"The abdominal organs did not show any noteworthy changes. The brain is somewhat congested, and on separating the anterior and middle lobes of each side, small miliary tubercles are seen in each Sylvian fissure. Near the origin of the left Sylvian artery the vessel is surrounded by a hard tuberculous mass, which almost obstructs it. On section, a tuberculous nodule of the size of a large pea is found in the left crus cerebri. It lies in the crusta at about the middle of its length.

The nodule is generally hard, but shows a few spots of caseous degeneration. A similar tuberculous nodule is found in the right crus cerebri, but is situated in the tegementum, and on a much higher level than the nodule in the left crus. The ventricular fluid is increased, and the choroid plexus injected.

"Several tuberculous tumours are found in the spinal cord. One is situated on the right side between the second and third cervical nerves; another on the left side between the seventh cervical and first dorsal nerves (Fig. 2); while a third is situated over the centre of the lumbar enlargement. The tumours seem to the naked eye to grow from the membranes, and to pass inwards in the cord. Patches of thickened membrane are found in many parts of the cord."

FIG. 2.

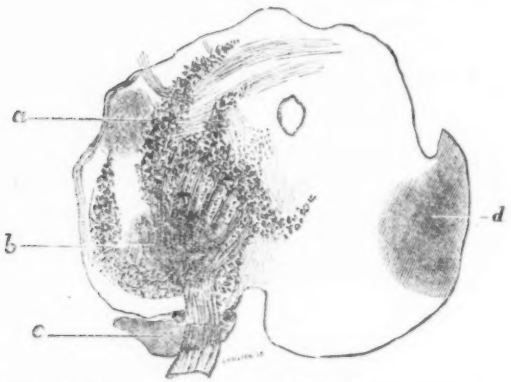


PORTION OF SPINAL CORD, SHOWING TUMOUR IN LOWER CERVICAL AND THE UPPER DORSAL REGION.

The microscopical examination was kindly conducted by my friend Dr. Larmuth, and the annexed diagrams are taken from sections of the crura cerebri (Fig. 3) and of the spinal cord on a level with the eighth nerve (Fig. 4). In the left crus (Fig. 3), a nodule of tubercle (*a*) is seen in the tegementum in such a position that it does not interfere with the third or fourth nerves; a second somewhat larger nodule (*b*), which involves the fibres of the third nerve and the motor tract, is situated in the crusta; while a third nodule (*c*) implicates the root of the third nerve after its emergence from the crus. This nodule was distinctly felt at the autopsy, although it is not mentioned

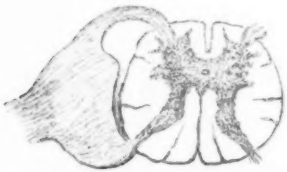
in Dr. Maguire's report. In the right crus a large nodule (*d*) is situated in the tegementum, but it does not involve either the third or fourth nerves or the motor tract.

FIG. 3.



(LARMUTH.) FROM A SECTION OF THE CRUS CEREBRI, SHOWING THE POSITIONS OF THE TUMOURS.

FIG. 4.



(LARMUTH.) FROM A SECTION OF THE SPINAL CORD ON A LEVEL WITH THE RIGHT CERVICAL NERVE, SHOWING THE RELATION OF THE TUMOUR TO THE NERVE-ROOTS.

In the section of the spinal cord the tumour is seen to be attached to the membranes over the posterior part of the lateral column, and to involve both the posterior and anterior roots of the nerve. The tumours consisted of lymphoid cells imbedded in a fibrillary matrix, but it was not thought necessary to search for bacilli, as the interest of the case centered in the localisation of the lesions.

Remarks.—My diagnosis of this case from the first was that a tubercular tumour was situated about the root of the third left nerve at its point of emergence from the crus; that a second involved the anterior and posterior roots of the seventh and eighth cervical and first dorsal nerves, and that tubercles

were scattered over the lumbar region of the cord, some at least of these being situated at the upper part of the lumbar region.

The slow and progressive manner in which the symptoms supervened, rendered it more likely that the lesion would be some kind of growth, and not a rupture or occlusion of a blood-vessel; while the presence of cicatrices, ankylosis of joints, scrofulous disease of the great toe of the right foot, and the evidences of an amputated leg, showed unmistakably that the growth would be tuberculous.

With regard to the localisation of the lesion, the fact that disease of the left third nerve was unaccompanied by disorder of the functions of any of the other nerves which pass through the cavernous sinus, or by hemianopsia from implication of the optic tract, showed that the tumour was not compressing the nerve in its course along the base of the skull; the absence of any evidence of hemiplegia of the right side of the body led me to believe that the nodule would not be situated in the crusta; therefore I concluded that it would be found at the root of the nerve after its emergence from the crus. It was thought at one time that there were slight evidences of paralysis of the third nerve of the right side, and this induced me to believe that we should find the tubercular deposit extending across the interpeduncular space to reach the root of the nerve on the right side. The evidences of paralysis of the right third nerve were, however, always doubtful, as it was probable that the restricted movements of the eyeball were due to the mental condition of the patient.

My anticipations with regard to the localisation of the tumour which caused the paralysis of the third nerve were not in all respects confirmed at the autopsy. It is true that a nodule was found at the point of emergence of the left third nerve, but I was very much surprised to find that another nodule was situated in the crusta, just in that position in which it might be expected to cause a spasmodic paralysis of the extremities on the opposite side of the body by interference with the pyramidal tract. Several reasons may be given for the absence of any evidence of right-sided hemiplegia of cerebral origin. There was no opportunity for examining the patient carefully after her discharge from the hospital on Feb. 9th, a month before her death, and it is therefore possible that a right-sided hemiplegia may have supervened in the intervening period. And even a certain degree of right-sided hemiplegia may have existed before the date of the patient's discharge from the hospital without having been detected, because the phenomena of a spasmodic paralysis would be

greatly masked in this case by the presence of other symptoms. The reactions of the tendons of the triceps and of the patella were abolished on the right side, owing to local disease of the elbow and knee-joints; the periosteal reactions of the radius and ulna would be lost partly from old-standing disease of the bones, and partly from a certain diffused atrophy of the muscles which even existed on the right side; and ankle-clonus would also be arrested from the diffused atrophy of the muscles of the lower extremity which was present; while a feeble grasp, and feeble movements of the limbs generally, might only be recognized as an expression of general weakness. But it is possible that right-sided hemiplegia had not existed at any time, for it is well known that a tumour of considerable size may encroach upon the pyramidal tract without causing paralysis, the fibres in such cases being pushed aside instead of being compressed or completely ruptured.

On observing for the first time that the third nerve and the hand were paralysed on the same side of the body, the first idea which suggested itself was that the case was an example of a direct instead of a crossed cerebral paralysis; but even a cursory examination sufficed to dispel this notion. The muscles of the left forearm, with the exception of the supinator longus, failed to react when their tendons were struck at the wrist, and these muscles with the same exception, as well as the intrinsic muscles of the hand, were found atrophied, and manifested the "partial reaction of degeneration" on electrical exploration. The hand also assumed the claw form which is met with in cervical pachymeningitis when the disease is on a level with the eighth cervical and first dorsal nerves, with the exception that hyper-extension at the wrist was not a marked feature of the case, inasmuch as the extensors, as well as the flexors of the wrist and fingers, were involved in the paralysis and atrophy. The implication of the extensors showed that the seventh cervical nerve was also involved in the disease, and it was therefore concluded that the lesion involved the motor roots of the seventh and eighth cervical and first dorsal nerves. The conclusion reached, from a consideration of the motor disorder of the left hand and forearm, was in the main confirmed by the sensory disorder observed, the anæsthesia being present in the area of distribution of the nerve of Wrisberg, the internal cutaneous, and the sensory branches of the ulnar, these nerves being derived from the eighth cervical and first dorsal nerves.

It will be remembered that the anæsthesia of the inner surface of the upper arm was much less complete than that of the corresponding surface of the forearm, which is explained by the fact that the former surface is supplied by the intercosto-

humeral nerve, which is derived from the second dorsal, the root of which was not involved in the disease.

Having once concluded that a tubercular tumour was situated in or near the crus cerebri; that a separate lesion, situated in or near the spinal cord, involved the anterior roots of the seventh and eighth cervical and first dorsal nerves, and the posterior roots of at least the last two of these nerves; and that the lesion was chiefly unilateral, there was no difficulty in inferring that the nature of the spinal lesion was tubercular; that it formed a more or less localised growth in the spinal membranes; and that the roots of the implicated nerves passed through it. These conclusions were amply verified at the autopsy. The existence of tubercles in the lumbar enlargement of the spinal cord was suspected from the presence of a diffused atrophy of the muscles of the lower extremities, and, in order to account for the incontinence of urine, it was assumed that some of these were situated in the upper part of the lumbar region.

The presence of the tumour in the spinal cord on a level with the third nerve was not suspected during life; but this is not surprising, when it is considered that the patient was unable to sit up in bed, and that consequently disorders of the innervation of the cervical plexus, difficult to detect under any circumstances, would be likely to escape notice.

The disorders of speech were attributed during life to interference with the function of the geniculate fasciculus of the pyramidal tract in its passage through the crusta; but we are now inclined to regard them as resulting from anæmia of the motor area of the left hemisphere, caused by the tubercular growth which surrounded the point of origin of the left middle cerebral artery.

CASE OF INJURY TO RIGHT SIDE OF HEAD, FOLLOWED BY COMPLETE PARALYSIS OF RIGHT ARM.

BY GEORGE THOMSON, M.D.,

Surgeon, Oldham Infirmary.

J. M., aged 34 years, was among a crowd watching a fire on the morning of December 19th, 1883, when the fire-escape, in passing up the street, came in contact with an overhead telephone wire, causing it to dislodge a quantity of masonry about twenty feet above the heads of the people. A heavy coping-stone was among the debris, and seems to have fallen on the heads of several men, severely injuring three, of whom J. M. was one. I saw him immediately after the accident, and found him suffering from symptoms of concussion of the brain, with a scalp wound about 1½ inch long on the top of the head over the right supero-antero-parietal area of the skull, which was denuded of periosteum to about the size of a shilling. No other injury to the head was apparent.

The early symptoms were quick, small pulse, cold surface, vomiting, and partial unconsciousness. When put in bed, he lay on his side with his limbs flexed, as in cerebral irritation. Towards evening, the pulse became slow (58) and the temperature slightly rose (100°), and the man passed into deep stupor, with equal pupils, slightly contracted, but reacting to light. His condition remained practically unchanged for two days, when (Dec. 21) he began to swallow a little. During the following night he had five or six clonic spasms, lasting a few minutes, beginning in the right face and extending to the right arm.

Dec. 22nd.—These spasms occurred several times during the day, but during the intervals he was more conscious, swallowing liquids freely, and addressing his sister once by name. There was no apparent paralysis of arm or face.

Dec. 23rd.—During the night the clonic spasm returned, accompanied by clenching of the hand and jaw, with cold surface and general collapse. This condition lasted about

three hours, and when it passed off, the right arm appeared to be completely paralysed. He became more conscious during the day, took liquids well; was aware of the presence of strangers, and put out his tongue when asked. The spasm recurred in the right face during the day, but not in the arm. The pulse was still under 60, and the temperature had fallen to normal. The leg was moved about quite freely. Dr. Ross saw him with me in the afternoon; and, notwithstanding the puzzling fact, that the paralysis and the external injury were on the same side, agreed that in case the circumstances became desperate it would be well to trephine where the skull was denuded of periosteum.

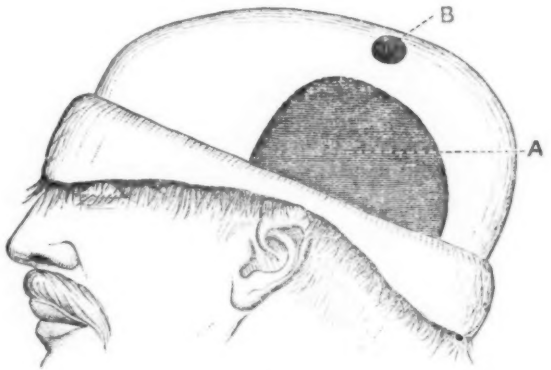


Fig 1.

Dec. 25th.—The pulse had gradually risen to 80, temperature 100°. He named people in the room, but had intervals of violent delirium.

Dec. 26th.—He was more continuously violent. Evening pulse 120, temperature 101°; pupils less contracted, and dilate widely when he is roused. Ice applied to the head.

Dec. 27th.—He had become so seriously worse, that I decided to try the expedient of trephining, in the hope of affording relief. I removed the circle of bone which was denuded of periosteum, and finding the dura mater very tense, incised it, liberating a considerable quantity of serous fluid. A slight relief seemed to follow, but he died during the day.

Dec. 29th. — *Autopsy.* — Assisted by Dr. Corns and Mr. Holton, I made an examination of the head. On reflecting the scalp, there was considerable extravasation of blood over

the left parietal surface, and also in the temporal muscles. It was noticed in removing the skull-cap, as it had also been in trephining, that the bone was unusually thick and tough. An extensive crack was found in the skull, describing two sides of a triangle, with the apex about $\frac{1}{2}$ inch in front of the scalp-wound. One ramus of the fissure extended forwards in a diagonal direction across the vertex, and terminated in the roof of the left orbit. The other ramus extended laterally, traversing the left supero-antero-parietal, infero-antero-parietal, and squamous areas, and terminating in the petrous portion in the floor of the middle fossa of the skull. External to the dura mater on the left side was a large venous clot (Fig. 1, A), apparently due to oozing from the diploë, and corresponding to the squamous, infero-antero-parietal, and infero-postero-parietal areas. The dura mater was lacerated for about $1\frac{1}{4}$ inch immediately under and in the direction of the fissure in the skull (Fig. 1, B), about $\frac{1}{3}$ inch to the left of the middle line and over the ascending frontal convolution. The cortical

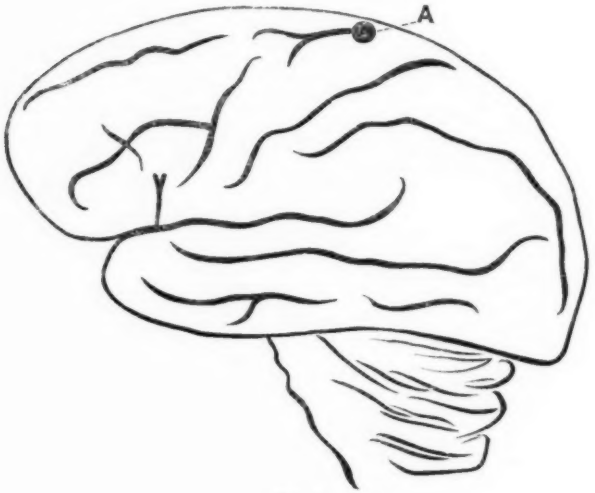


FIG. 2.

substance of this convolution was also lacerated for about $\frac{1}{3}$ inch immediately underneath the tear on the dura mater, and contained a small clot about the size of a pea. (Fig. 2, A.)

Remarks.—The most striking feature of this interesting injury, is the fact that paralysis was present on the right side,

while the injury, so far as evidence was forthcoming before death, was on the same side. In point of fact the paralysis was the only reliable indicator of the portion of brain injured, the marks of external injury being quite misleading, as the post-mortem appearances show. It is certainly very unusual and extraordinary that so extensive a fissure of the skull should bear such relation to the situation of the blow, and that the point of injury to the brain and its membranes should be neither at the site of the blow nor the point of contre-coup, but at a point on the left side nearly corresponding to that on which the blow fell on the right.

Critical Digest.

ON VERTIGO.

BY GEORGE PARKER, M.A., M.D. (CAMB.)

VERTIGO, or giddiness, is a symptom that almost every one has experienced under some circumstances or other, and often under such widely different conditions, that one is tempted to believe there can be no common cause. We are met at the beginning by the difficulty, that it is a purely subjective phenomenon, and therefore out of the range of many means of inquiry. We can, however, cross-examine our consciousness on the one hand, and on the other register the physical signs correlative to giddiness, such as certain forms of inco-ordination and forced motion. That they are correlative seems admitted on all hands, nor is this negatived by the fact that either may occur without the other. For ideas of action are not necessarily carried out into effect, and the mental states producing action or produced by sensation need not rise into the sphere of consciousness. Vertigo, then, may be closely related to certain movements, though it does not always accompany them.

It varies from a mere sense of confusion, which perhaps ought not to be included under the name, to (*a*) a sense of all things moving about me; or (*b*), my moving while they are fixed; or (*c*), finally a definite sense of forced motion in a given direction, which motion is actually carried out. It is not every sense of motion which produces it. If I leap, jump down a hole, dive, or fall off a chair, there is a sense of motion; but giddiness does not necessarily appear, it seems to involve a sense of abortive effort.

If then it correspond to a *negation*, a *failure* of action,or co-ordination, can we get any definite result by introspection or by judging the physical result?

We may not be able to make clear that which in itself is confusion or failure to act; but its cause may still be discovered, and it may itself be compared with the normal action, of which it is the distortion.

Vertigo is generally defined as "the consciousness of disturbed locomotor co-ordination," or, as Dr. H. Jackson says, "a rudimentary disorder" of the same; or if, with Ferrier and Goltz, we distinguish the allied functions of equilibration and locomotor co-ordination, it is the consciousness of disturbance in equilibration.

It implies a loss of proportion between the efforts to maintain the erect posture, whether real or ideal.

This generally depends on a conflict of impressions, clearly not of all kinds, for giddiness does not follow if one hand reports heat and another cold. Nor do the conflicting impressions of the insane make them necessarily giddy, nor does a contradictory logical proposition produce it. It seems that at least four senses—touch, sight, the muscular sense, and the sense of equilibrium, perhaps also that of hearing—are employed in maintaining the erect posture; and if one of these contradicts the others, inco-ordinated impulses may be sent out, from which giddiness follows. Thus a person feels giddy on looking over a precipice; for the sight of the void beneath contradicts his muscular sense, and irregular efforts follow producing giddiness.

Again, without any impression upon a sense-organ, but by acting on parts of the central organ concerned through electricity or disease, there results failure of equilibrium and giddiness. Thus vertigo is not, I think, directly the correlative of conflicting sensation, but of abortive action, and the closeness of the connection is shown by the fact, that when giddiness is very great and actual motion appears, it always is in the direction which the whirl of the giddiness took.

I shall try to show that the vertigo of ordinary clinical cases depends on distortion of normal impulses, and is caused by injuries to different parts of the same machinery, i.e. of equilibration.

The diseases in which giddiness occurs are endless, and the question, "what can be the normal functions thus interfered with in so many directions and resulting thus in giddiness?" would lead us, in the absence of other proofs, to expect a *constantly* used mechanism, such as that for maintaining equilibrium.

To commence with a frequent cause, going to the root of the matter—*lesions of the cerebellum*. There are a great number of cases on record, of injuries, growths, and degenerative changes in this organ, followed by vertigo. Though its functions are not quite clearly determined, it seems on the whole to act as a great motor centre; sometimes from unfelt impulses, as the centre of a reflex chain, and sometimes at the[1] instigation of the more complex changes which are accompanied by consciousness, and in conjunction with consciously directed movement. Finally, it has, among

[1] Three difficulties may be mentioned in fixing the functions of the cerebellum. 1. Irritation may spread to other organs, such as the cerebrum. Brown-Séquard even maintained that the cerebellum is not the agent for the co-ordination of movements at all, and that we must look elsewhere; but this view of his finds few supporters. 2. The possibility of the other half taking on the whole work when one lobe is disabled. But this, though true of the cerebrum, seems opposed to the mode of action in cerebellum. 3. The possibility of the cerebrum being substituted for it when diseased or injured. What is its normal function? Bastian insists that it is the supreme organ for the reinforcement and regulative distribution of the outgoing currents, not only for co-ordination. Luys, that it engenders and distributes the force needed for movements. Flourens, Longet, and others, that it co-ordinates. Broadbent regards it as co-ordinating movements guided by vision, which subserves it on one side as the muscular sense does on the other.

other functions, that of co-ordinating distant and unlike movements of the neck, trunk, and limbs, which maintain our balance.

To consider the *forced movements* seen in some diseases, or artificially produced—while removal of the deep layers of the cerebellum produces general loss of co-ordination without motor paralysis, section of the middle peduncle gives rise to forced rotation round the longitudinal axis, and to nystagmus. Now the first is seen in some diseases, where the patient has severe giddiness; and the second will of itself produce giddiness, as in some eye-complaints. Again, injury of the anterior part of the middle lobe is followed by rolling forwards; of the posterior, backwards; of the lateral lobe on one side only, by rolling sometimes to that side, but sometimes to the other, which may depend on interference of conscious action. So, too, disease of the vermiform process causes a rush forwards; and generally, in tumours and hæmorrhage in the cerebellum, there is staggering and giddiness of a continued non-paroxysmal type. If in disseminated sclerosis one of the patches should affect this organ, giddiness occurs, which, Charcot says, is generally gyratory; the patient and all objects seem to revolve together. This occurs in three-fourths of the cases of disseminated sclerosis, but must not be confused with that caused by the nystagmus present. If a galvanic current be sent through the mastoid fossæ from right to left, giddiness follows, the head and body sink towards the right, and objects seem to revolve from right to left. If the eyes are closed, all support beneath the left side seems gone at the closing of the current. The eyes too move towards the right, and these same events occur if the actual cerebellum be stimulated. Again, if we are turned round rapidly from right to left, objects seem to fly from left to right; as this produces giddiness, we bend our body, and turn our eyes to the right, to neutralise the sensation. This may explain the galvanic results, showing that the right side of the cerebellum co-ordinates the mechanism, which prevents displacement of equilibrium to the opposite side, and *vice versâ*.

Yet occasionally the entire cerebellum has been removed without obvious loss of equilibration, and Dr. Oliver gives a very instructive case of a tubercular tumour in the left lobe, with vomiting and periodic headaches. The young woman, however, played the piano, and *walked about* day after day, a short time before her death. He suggests that here was a substitution of the cerebrum, and conscious adjustment for automatic; that she reverted to the condition of a learner, making the co-ordinations necessary, as she had done at a time when they had not become automatic.

Weir-Mitchell found that pigeons recovered in the course of some months their co-ordinating powers after extirpation of the cerebellum, by apparently relearning the processes.[1] So too in the case of a girl recorded by Cruveilhier, where the cerebellum was atrophied. Tuke mentions several other cases of atrophy without inco-ordination. After all, we know that we can replace automatic action by conscious, when the former has broken down. Thus in

[1] The exercise of this conscious balancing caused them great fatigue and exhaustion owing probably to the constant attention required.

crossing a plank, we can, by an effort of mind, force ourselves to stand firm; the drunkard pulls himself together with an effort, after his automatic power is gone.

It may be said, that the cerebellar disease in any of these cases merely caused an irritation elsewhere; but there is no other such place discovered, the lesions causing great giddiness and inco-ordination appear often too small and chronic to affect, for instance, a distant part of the cerebrum, and in any case the cerebellar lesion is a link in the chain of causation. It may be necessary to remark here, that while co-ordination does appear to depend on the cerebellum, we have no reason to think that giddiness can occur without cerebral charges. Co-ordination may go on after the cerebral lobes are removed, and therefore, when consciousness has ceased, and giddiness has become, so far as we know, impossible.

Though irritation of the cerebellum produces giddiness, I do not for a moment suggest that it is a sign of cerebellar disease only, for it may accompany almost any brain-disease, though indeed most usually associated with cerebellar. To take the 73 cases of abscess in the brain which Sir W. Gull gives. I find, upon analysing them, only one case of vertigo which could be traced to cerebellar disease, out of 8 in which it was present. Again, Dr. Bradbury refers to a pyæmia where sudden vertigo came on, and the *post mortem* revealed plugging of the middle cerebral. Still all these cases may be looked on as indirect stimulation of the cerebellum through the associated tracts, such as the optic lobes; there is nothing in them contradictory, at least, of Ferrier's results.

How does an affection of the brain act so as to cause vertigo? The answer usually made is—By disturbance of the cerebral circulation, stimulating the nerve matter to irregular explosions. And here, after the analogy of respiratory convulsions, which are produced by deoxydised blood, acting upon one spot alone, we should expect only that definite area to be acted on where we can produce like effects by puncture or stimulation. Positive proof is wanting, because it is difficult to isolate part of the circulation;[1] but I hope to say more about this, later on. Again, how does the circulation affect the brain-substance? This would seem almost as crude as the old theory, that rotation caused vertigo by dislodging bits of brain substance, unless we have a specialised organ in which changes may be induced. Such an organ does exist and produce vertigo, both under disease, and when we experimentally alter the tension of the fluid in it. This tension, in turn, is held by many authorities to vary with the blood-pressure.

The next class of diseases is *the auditory;* and here we find much that will serve to explain the real nature of vertigo. It is a common experience that, in examining the membrana tympani, some degree of giddiness is produced, especially in syringing or if

[1] Dr. Bradbury's case, quoted above, comes near doing so, but the vertigo may have sprung from the pyæmia and endocarditis long before the plugging took place. Cases where vertigo follows on pressure upon the carotid may depend on interference with the cardiac depressor nerve, as in Czermak's case, or on compensatory dilation of the vertebral.

the ear be blown into. Again, the pressure of wax causes sometimes most severe vertigo; and Hughlings-Jackson describes a case when a powerful soldier was rendered almost incapable by a louse scratching on the membrane.

Still more serious are the occasional effects of a catarrh, or inflammation of the middle ear or Eustachian tube, and by which the stapes is pressed in upon the foramen ovale. But the most important case for us is the condition in Ménière's disease. A healthy patient is suddenly attacked by fits of tinnitus, vertigo, nausea and vomiting, but without loss of consciousness. He tends to fall towards the affected side; the attacks become more and more frequent. A patient, whose case I examined day by day, complained first of a sudden pain in the head, as if crushed under weights; next, giddiness, when the ground seemed to rise up on his right side. He could hear a watch 2 feet from the left and at not more than 2 inches from the right ear. After his paroxysmal exacerbations, as Charcot calls them, he often falls into a sweat. The usual course is that, after some years, the noises and vertigo cease, but the patient becomes stone-deaf. There is in the true Ménière's disease, an invariable coincidence of vertigo, tinnitus, deafness (especially perosseous), with more or less of nausea and syncope; and occasionally there is nystagmus, discoloration of the hands, and stiff or wry neck, but no other sign of disease.

The tinnitus is much complained of, and rises often to a shrill scream before a paroxysm. As to the ætiology, it seems pretty certain (1) that disease of the membranous semicircular canals is the cause of the symptoms; (2) that a definite brain area is affected beween which and the canals there is connection by continuity of nerve-fibres only; and (3) that these fibres are distinct, may really be considered as a 13th pair, and have nothing else to do but to form this connection. For not only is there found on dissection an inflammatory exudation, more or less solid, in the canals, and (as if to mark the identity of its mode of action with cerebellar stimulation) not only is there a tendency to fall in opposite directions, according as the lesion is irritative or destructive, but it has been pointed out that the group of symptoms, minus vertigo, do not mean disease of the canals at all, but of the middle ear and cochlea,[1] and that vertigo again depends on certain fibres of the 7th pair, which run from the canals to the superior nucleus and then to the cerebellum. Indeed, it would be contrary to analogy for such a nerve of special sense as the real auditory to produce anything but its own sensation of sound. Tinnitus, deafness, and what Dr. Jackson terms the "vital" effects, such as syncope, depend on the other or true auditory fibres running from the cochlea to the inferior nucleus in medulla. Since this nucleus is close to those of the vagus and spinal accessory, the vital effects can be accounted for as an overflow from one nerve to another, in so far as they are motor, or as a transference (like that of hip pain to the knee), if they are sensorial.

[1] Macbride, 'Guide to the Study of Ear Disease,' p. 149. Cochlitis alone produces no giddiness, though some forms of it frequently follow affections of the middle ear.

This is confirmed by the fact that vomiting does not occur in mild cases, and is always subsequent to the vertigo in auditory disease, since the nervous discharge must be of a certain strength to overflow its track, which in any case would be first affected.

Here then is an organ for the production of vertigo as definite as that of vision. Can we account for all stomach and circulatory vertigoes by its means?

Let us examine Dr. Woakes's theory. While he accepts the ordinary explanation of vertigo in Ménière's disease, he teaches that the paroxysms are caused by irritations in the heart, stomach and arm. These are conveyed by the inferior cardiac nerve, and by branches from the brachial plexus and vagus, to the inferior cervical ganglion, which sends filaments, probably vasomotor, to the vertebral artery, and this in turn supplies the semicircular canals by one of its branches, and regulates the fluid in them. This well explains those cases in which symptoms of Ménière's disease exist without any organic ear-affection, while the accompanying fainting and nausea are primary, and the causes rather than the effects of the vertigo. Where a lesion of the canals exists, such a shock from the viscera will reinforce it, causing the paroxysmal "fits," as my patient used to call them. Of course these paroxysms could be accounted for by changing conditions of the nervous system, or by the so-called summation of stimuli. There is no special means of explaining by Woakes's theory why vomiting should precede the vertigo, when stomachal and not auditory, and should follow it when of auditory origin, and this the radiation doctrine does.

He illustrates the effect of the nervous chain by certain shot-wounds of the brachial plexus which occurred in the American war. Patients were observed on receiving them to fall suddenly to the ground without loss of consciousness, and probably from changes in the canals.

The great interest to us is that it so well explains vertigo *à stomacho læso*, and the forms which occur in heart-disease, and after many poisons where no lesion of the canals exists. He would even exalt vertigo to the rank of a beneficent function, intended to lay men in the best position to recover from faintness.

Again, he illustrates the reflex action of the ganglion by noting the congested hands seen in tobacco-poisoning; but this congestion occurs in Ménière's disease, and its occurrence discredits his argument. For though dilatation of the vertebral may be governed by the inferior cervical ganglion, it is hard to see how changes in the canals or vertebral can affect the ganglion, and through it the vessels of the arm. Dr. S. Mackenzie is my authority for the fact. I have not, I regret, yet noted a case in the aural department at St. Bartholomew's.

After all, there is no crucial experiment by which Woakes's theory can be tested. Who has stimulated the inferior cervical ganglion and produced vasomotor changes? The nearest approach to it is Dr. Chapman's water bags, where, as he asserts, he can stop bleeding from lungs or nose by hot-water bags to the cervical spine, and

cause an afflux to the head by applying ice bags to the same place. I need not do more than refer to Claude Bernard's analogous experiments.

Again, how can we limit the effects to such a definite area as the labyrinth, or how do we know that an increased blood-pressure affects the fluid in the canals? I suggest that the auditory artery alone of the branches of the vertebral runs through minute bony channels in conjunction with a nerve, and that dilatation of the artery causes pressure on the nerve.

Finally, Woakes does not throw any light on the vomiting and fainting which accompany constant vertigo in the most severe form of Ménière's disease, where no heart or stomach trouble exists.

Of course it is difficult to prove such a negative as this non-existence, but it seems to me that we cannot drag in a stomach or heart irritation in every case.

Certainly if we regard the theory as merely one of several possible ways in which giddiness can be brought on, it explains Dr. Foster's difficulty, that there is often disease of the canals without vertigo. For if we are inclined to admit, that there is a centre for equilibrium like those of respiration and vomiting, and that it is to a great extent dependent on the semicircular canals, then Foster's case admits of solution in two ways. First, the disease may be so mild as to produce no vertigo without helping current from the inferior cervical ganglion, and this with healthy organs may not occur. Secondly, the other senses that contribute to equilibrium may be in some cases sufficient to replace the aid given normally by the canals. In an early case mentioned by Russell Reynolds 30 years ago, a Dr. Wollaston describes a vertigo which used to seize himself on rising from a bent posture, but *only* when he was fatigued or weakened. Whether he had really ear disease or not, the vertigo only came when the central organ was in an abnormal state.

Foster mentions, though he doubts the correctness of the observation, that vertigo can still be produced by rotating an animal after section of both auditory nerves, arguing that sensations from the auditory canals cannot be the sole cause of this kind of vertigo. This experiment, however, disagrees with an important one by Dr. James, of Harvard. He examined a number of deaf mutes to see how far vertigo could be caused in them by rapid rotation. Out of 200 healthy students of Harvard taken for the purpose of comparison, only one was free from giddiness after rotation in a swing with the eyes closed: whereas of the deaf mutes (519 in number) 186 remained perfectly free from the feeling, and 134 were only slightly affected. Unfortunately the actual lesions cannot be ascertained in all cases of deafness. Another curious fact was, that of 15 deaf mutes who had been to sea, only one had suffered from sea-sickness. To sum up: inco-ordination is rarely seen in congenital deafness; it ceases in Ménière's disease with the subsidence of inflammation, unless, as some physiologists say, the cerebellum has been involved by an ascending mischief; and I think we must

confess, after Dr. James's experiment, that there is no reason to think vertigo can be caused after destruction of the auditory nerve by anything like rotation which does not affect sight or the muscular sense.

Before going to the other diseases in which giddiness occurs, we will pause to consider the normal action of the canals. I do not know how we can escape the conclusion which has been felt since the time of Flourens, that they concern the sense of equilibrium and direction in space. Passing by the stories that the savage huntsman is able to find his way by some such sense of direction, there is much evidence that when one is placed blindfold on a rotating table, it is possible to judge of the direction and angle gone through. Moreover, and this is of interest to us, if in a healthy pigeon they be cut through,

(1) It moves its head from side to side constantly.

(2) It loses the power of co-ordinating movements.

(3) It has not paralysis, uncontrollable impulses, or hallucinations of sight or hearing, as far as can be judged, but seems simply "dizzy."

(4) If a rabbit be operated on, there is also nystagmus.

(5) If only the superior vertical canal be injured, the animal falls forward; if the post-vertical, he falls backwards; and if the horizontal one, to the side of the injured canal.

There is a striking parallel to the results obtained from the cerebellum, and shows the closeness of their connection, whether we believe the canals originate for us the idea of space in three dimensions or simply register movements. Indeed I am hardly now concerned with the question apart from the vertigo of rotation. It is said that if the canals be drained very slowly, no vertigo is produced, and vertigo in Ménière's disease ceases with the active inflammation of the canals. Thus we may conceive of them as serving no purpose normally whatever towards equilibration, and yet, from their intimate connection with its especial organ, as capable of collecting from various parts and transmitting to it shocks sufficient to disturb it profoundly. Just as the wire of a battery placed in my mastoid fossa conducts disturbing currents, though not a normal part of my organism, so sudden section or quick rotation may still stimulate the canals to the production of such disturbances.

We must now pass to other clinical forms; but as we have been compelled to mention *Vertigo à stomacho læso*, as Trousseau calls it, we may consider it for a few moments. Some warm morning it happens that a man engages in business for 2 or 3 hours after a hearty and rich breakfast. All at once his sight becomes dim, he finds himself very giddy; he may even faint outright, but things generally seem to revolve around him, till he is relieved by copious vomiting. Subsequent attacks may occur without, as Trousseau thinks, any discoverable indigestion or excess of diet.[1]

[1] It is doubtful indeed whether the stomach is at all the seat of the real lesion in these cases. Thus Trousseau remarks, "I have frequently asked myself whether the treatment which apparently affected the digestive organs was not, unknown to me, addressed to the nervous system."

The vertigo is even more sudden, violent, and intermittent than in Ménière's disease, and the prostration great. If there is no indigestion, what causes the later attacks? Now it happens that vertigo occurs often in migraine, and though it is here usually referred to preceding sight and touch-affections, the migraine itself is frequently defined as caused by vasomotor changes (shown by the turgid arteries, &c.) which are produced by so called nerve-storms. Migraine, says Bristowe, has a close pathological relation to epilepsy, and, I think we may add, it is closely related to certain vertigoes.

To look at it in another light. Besides the suggestion of Trousseau that this stomachal vertigo is purely a nervous affection, there are other facts pointing to some intimate connection between the cerebellum and the stomach. Cyon, by injuries to the canals, produced vomiting; so too it exists in cerebellar or Ménière's disease: irregular movements cause sea-sickness: stimulation of cerebellum (Schiff) causes peristalsis; in typhoid there is again notable vertigo; and lastly, Ferrier oddly remarks, that in the felidæ, where equilibration is singularly perfect, there is a great development of Pacinian corpuscles in the mesentery. Whether we accept Woakes's or H. Jackson's theories, the number and variety of irritations in the vagus area with which vertigo occurs is startling.

Laryngeal irritation in bronchitis and emphysema, and even from polypi, is, according to Dr. Gee, a powerful excitor of vertigo. The patient's face is livid and turgid; he becomes giddy 15 or 16 times a day, and presents a convulsive movement of the arms, which recalls their discoloration in aural disease. Can there be a vasomotor change affecting the canals? clearly not through the inferior cervical ganglion. (*a*) Charcot suggests the superior laryngeal. The vagus itself being in close relation with the lower peduncle, may transmit through it a current to the equilibrium centre. (*b*) Or if we remember that irritation may spread up the mucous membrane to the Eustachian tube, the case becomes an aural vertigo. (*c*) Lastly, the sympathetic branch from larynx passes to the superior cervical ganglion, which gives off a branch to the external carotid, explaining the turgidity of face, and others through the carotid plexus to tympanic plexus and fenestræ, while the tensor tympani is reached by the small petrosal.

In *influenza*, and perhaps in mumps, we find exceptionally great tendencies to vertigo, and these are just the two diseases in which the invasions of the Eustachian tube are very common. Here again we need not ascribe it to general disorders of the brain-circulation, but to hyperæmia of the middle ear.

Anæmia is one of the most common antecedents of giddiness. Not only is loss of blood a cause, but various morbid conditions which are (from their frequency) perhaps more interesting to the physician than the local maladies we have been considering. To my mind this giddiness is a most mysterious matter, though we are apt to think of it as only the confusion arising from weakness. Yet why should the man who is suffering from anæmia feel himself or his surroundings *move* in either direction? It is very different from the faintness or confusion that flow from his weakness. We have

seen reason to associate vertigo in many cases with an irregular outpour of energy from the cerebellum in certain directions. Can the results of anæmia be thus explained? (1.) In most cases of anæmia there is an alteration of the pressure of the blood. This can act through the canals on the cerebellum, just as tapping them does. (2.) As the deficient or abnormal blood causes a greater tendency in the nerve cells to explode, we get convulsions and failure to maintain equilibrium, because the efforts or discharges are now unmeasured and irregular. In either case we are acting on *the organ* of co-ordination, and produce in it a rudimentary and imperfect action, which is reflected in consciousness as vertigo, or a special form of motion.

Not only in chlorosis, and pernicious anæmia, hæmorrhages, amenorrhœa, menorrhagia, and heart-disease does it occur, but also on leaving a chamber of compressed air, or after the sudden loss of cerebro-spinal fluid.[1] But here we meet with a difficulty. Why does not vertigo occur every time we fall asleep, since the brain becomes anæmic then? The diminished blood then clearly does not cause explosions more numerous than usual. We die our daily death without a moment's giddiness, so that *changes in the circulation of the brain itself cannot be of much importance*, although those in the general circulation are. Thus we are again led to the stimulation, not of the whole brain, but of some end organ, possibly the canals.[2]

The action of therapeutic agents and poisons agrees singularly well with these results. The depressants are remarkably apt to cause giddiness, and by these, as Dr. Fothergill says, the tone of the arterioles is so relaxed, that the patient is actually bled into his own body. Then, again, nitrite of amyl, though uncertain in its effects on different people, very often causes giddiness by this relaxation of vessels. In opium, alcohol and carbolic acid, both results are produced; but there may be a doubt whether the effects of belladonna are not produced indirectly through distortion of vision. There is some evidence for the opposite view, such as the flushing of the facial capillaries. The dryness of the throat may point to pneumogastric changes. The giddiness of tobacco-poisoning is not less marked than that of what Woakes calls its antidote, camphor; and if we examine Böhm's classification of drugs affecting the circulation, we get a pretty complete list of those causing vertigo.[3] Now there is one drug, chloral, which Ringer

[1] Thus Magendie produced staggering and apparent giddiness in a dog by puncture and withdrawal of the arachnoid fluid.

[2] But will not sleep-anæmia entail stimulation of the canals? Not of necessity, for there is no evidence, as far as I am aware, showing that the arterial contraction extends to the auditory artery, and if it did, the simultaneous quieting of the cerebellum would counterbalance any message from the canals to it.

[3] Böhm's list (abridged), 'Brit. Med. Jour.,' January, 1881. "Drugs acting on circulation:"

1. Those acting on heart direct, and when removed from body, causing increased energy; *e.g.* digitalis, helleborein, veratria, physostigma, camphor.
2. Those acting on heart direct, and when removed from body, causing enfeeblement; *e.g.* copper, zinc, tartar emetic, apomorphia (?)

and others never mention as causing it, nor do I recollect an instance myself, and yet it at once causes anæmia of the brain. This perplexed me a good deal at first; but on referring to the experiments of Bouchut, Andrews and Anstie, we find that in ordinary doses the pulse is not affected, and that, if anything, there is no fall, but a slight initial rise of blood-pressure, and even this is not beyond a doubt. That is, in a clear case of change in cerebral circulation there is no vertigo. Dr. Moxon has given strong reason for thinking that very few of the changes in the general circulation, which are frequent causes of giddiness, affect the cerebral.[1] The most constant symptoms of an overdose of gelsemium are giddiness and dimness of sight, with little or no effect on the blood-pressure, the mode of action probably being through injury to the optic centres which are in communication with the cerebellum. The vertigo is increased by motion, and is apparently subjective, *i.e.* of the patient going round, as in cerebellar lesions. This, I think, affords a contrast to poisons acting on circulation, and to excentric vertigo generally, and Ringer finds it gives relief in Ménière's disease.[2]

In chronic alcoholism a rare form of vertigo appears, in which objects seem turned upside-down. This has been also observed in gout. In ordinary drunkenness there is paralysis of vasomotor centres and of the muscular sense, the vertigo being generally objective.[3]

In Ringer's list of diseases, where high tension is present, almost

3. Excitors of (*a*) automatic centres, (*b*) inhibitory ganglia; *e.g.* (*a*) alcohol, chloroform, chloral cyanogen; (*b*) muscarin, nicotin.
4. Excitors of both; *e.g.* atropia, and (?) nicotin.
5. Stimulants of cerebral centres of cardiac nerves; *e.g.* prussic acid, amylnitrite, CO, irritating gas in throat.
6. Stimulants of walls of vessels; *e.g.* tannin and astringents generally.
7. Stimulants of vasomotor centres; *e.g.* (?) ammonia, strychnia, picrotoxin.
8. Paralysis of vasomotor centres; *e.g.* ether, chloroform, chloral, alcohol, aconitia and irritating gas.

[1] He lays stress on a self-regulating mechanism, by which the veins are locked when the supply is reduced; on another, by which the turgid choroid plexus presses laterally on the vagus trunk, and on the flow in and out of the cerebro-spinal fluid. He shows that enormous atmospheric pressure has no effect on the brain, and that epileptic convulsions cannot depend on brain-anæmia produced by vasomotor changes; for while these only reduce the size of a vessel $\frac{1}{4}$, and in the long time of from 4–13 seconds, Kussmaul could never produce convulsions, if even one out of the four arteries to the head were untied. It is true he regards vertigo as caused by changes in the cerebral circulation, but his arguments on convulsions really support my view that vertigo cannot be so caused.

[2] For comparison with Böhm, I subjoin the chief poisons causing giddiness, as given by Guy and Ferrier.

Narcotics: opium and morphia. *Deliriants:* Belladonna and camphor, &c. *Inebriants:* alcohol, carbolic acid, nitrite of amyl. *Convulsives:* (?) strychnia. *Heart depressants:* tobacco and lobelia. *Heart asthenics:* HCy, aconite, digitalis, colchicum. *Asphyxiants:* CO, CO_2, and H_2S.

[3] Anstie gives as the order of phenomena: The vessels are contracted, then relaxed, with paralysis of the vasomotor centres and cord and loss of muscular sense. Intellectual confusion, paralysis of respiratory centre, and lastly of heart follow. The blood is charged with alcohol, which stops oxygenation, and the nerve substance is paralysed, for when a living nerve is surrounded by alcohol it ceases to conduct.

every one is marked by the presence of vertigo; in degeneration of vessels, kidney disease, gout, jaundice, lead-poisoning, ergot and gallic-acid poisoning, fevers, &c., this is the case. There seems to be only one common property in these widely different conditions, viz. change of blood-pressure, which would readily act on a cerebral artery where there is a bony channel, together with hardly any anastomosis, and an end-organ easily stimulated by pressure.

In fevers there is often an accumulation of fluid in the middle ear, which requires no explanation.

The action of quinine and salicine in producing giddiness, though well marked, is so little understood, that I can make no use of it.[1]

In epilepsy there are two varieties of vertigo. In the *grand mal*, 77 per cent. suffer from prodromal attacks of it. This is always of falling towards one side,—never, as in brain-disease, of spinning round, nor as in excentric vertigo, of the room moving. The *petit mal* frequently consists solely of attacks of giddiness. Besides these cases, epilepsy is often brought on by ear-disease, and then paroxysms of vertigo are a marked feature.

How is it caused? Dr. Moxon speaks of it as produced by the momentary stoppage of the heart, which he thinks occurs in each fit. This might act by radiation from vagus to 7th or (through altered pressure) on the canals. But the vertigo occurs often at a very early stage, and must therefore precede heart changes. Thus, as to the brain anæmia said to exist, Bristowe argues that it may be a co-effect and not the cause of the tonic convulsions. In the general character of this vertigo there is reason to think it differs from the last class, and is caused either (1) by altered sensations, or (2) if, with Reynolds and others, we think that the beginning of an attack consists in a sudden explosion of nerve-cells affecting the medulla, and causing convulsions inhibiting the heart's action, then the early giddiness would be merely an extension of this wave of disturbance to the cerebellum and auditory nerve.[2]

In sunstroke, vertigo occurs in one only of the three forms. It is not met with in the syncopal or asphyxial types, according to some authorities, but only in the hyperpyrexial, which is also marked by epileptoid convulsions.

[1] On the one hand it is asserted by Kirchner that quinine produces congestion of the labyrinth; but, as Dr. Moxon has shown, post-mortem appearances are very deceptive in these matters. Others again consider it causes anæmia. Moreover, Woakes was led to discover that hydrobromic acid cures the vertigo of labyrinthine congestion by an observation of Dr. Fothergill that it prevents the aural troubles of quinine. This, of course, supports Kirchner's doctrine.

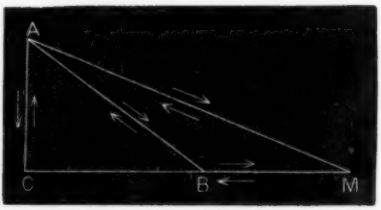
[2] It has been objected that if vertigo is, as I suppose, an affection of one organ, the cerebellum, in every case, why is it sometimes the sole symptom of epilepsy for long periods, alternating with rare attacks of the grand mal, and in other cases similar vertigo has no connection with epiplepsy whatever? Even when no other symptom is visible, there is probably in the epileptic vertigo an unperceived epileptic change. This is the foundation of the whole, and its effects may overflow the organ of equilibration or not; it has this alternative loop to traverse, but this loop or organ is equally affected by non-epileptic causes.

In locomotor ataxy we have a failure of co-ordination rather than of equilibration, and though these react on each other, so that loss of motor co-ordination renders equilibration impossible, or *vice versâ*, still a distinction can be made. As loss of equilibration alone is accompanied with vertigo, the latter affection is by no means so frequent in tabes dorsalis as might be expected. In an analogous affection, a man whose leg is "gone to sleep," as it is called, may stagger without any vertigo.

It will, however, be useful to consider this parallel disturbance of co-ordination. While the centres for real co-ordination are in the mesocephalon, the apparatus for combining a few muscles together for one act lies to some extent in the cord. Both may be involved. There has been a very great discussion whether the sensory part of the mechanism is at fault, or the central, or the efferent.

To me it seems that Erb's view is satisfactory, that it is a motor affection, that the co-ordinated stimuli sent down to maintain any complete act are cut off in their descent by lesions in certain unknown tracts in the cord, and that the inco-ordination cannot be accounted for by the anæsthesia, though it may be increased by it. In the Spaeth Schüppel case,[1] complete spinal anæsthesia existed without ataxia: the man walked, ate, and dressed himself, as long as his eyes were open, perfectly. In other cases complete ataxia existed without any anæsthesia. The often discussed tottering in the dark depends on cutting off the sensory impressions which might assist conscious co-ordination.[2] The essence of the disease is failure to co-ordinate and not failure of sensation.

When vertigo exists, it arises from (1) the accompanying diplopia, strabismus, and (2) the conflict of sensations produced by partial anæsthesia. Generally, though there is inability to co-ordinate for movement, there is power to maintain equilibrium, as far as the unmanageable limbs can be governed; there is a determination of what is needed, but not power to move the limbs as desired. We may represent these results diagrammatically, thus:—



A. Centres of consciousness (sensation and volition). B. Co-ordinating centres. C. Equilibrating centres. M. Muscular system. The arrows indicate that the tracts are everywhere double, and consist of both afferent and efferent fibres.

In tabes, B is cut off from the muscles, and reeling occurs. There may or may not be an affection of sensation, but there is nothing

[1] Ziemssen, xiii. p. 88.

[2] See "Controversy on the Theory of Locomotor Ataxy," in 'British Medical Journal,' Dec. 6, 13, 27, &c.

to cause giddiness in B's being cut off. Now if C be irregularly stimulated by A, or receive contradictory reports from the senses so that it acts irregularly, its effect on the centre A will be represented in consciousness by vertigo, and B will be misdirected, so that true vertiginous reeling occurs.

As we have said, reeling may occur when A is destroyed; *i.e.* C can act on B without affecting A.

Finally, it is worth noting, that if C be cut out of the system altogether, co-ordination can go on consciously by A alone, or by A and B, as in the pigeons whose cerebellum was removed.

Sight sensations must be very briefly considered, though their importance is great. The superior peduncles of the cerebellum are found to begin at the red nucleus just below the corpora quadrigemina, probably bridging the gap between the optic tract and the organ chiefly concerned with equilibration. Some authorities would trace a connection in the substance of the valve of Vieussens itself. In practice we find an immense number of vertigoes caused by eye-affections. (1) Myopia, nystagmus, paralysis of sixth or third nerve, and amblyopia are instances. G. Stewart gives a case of minor nystagmus, where the giddiness was stopped by pressure fixing the eye, when all other treatment failed. When one eye is *suddenly* destroyed, rotation takes place, because there is inequality of stimulation to the centre of equilibrium. (2) Looking down a precipice is one of the most common causes, and here the impressions received by sight contradict those from other senses; touch, the muscular sense, &c., by which we ordinarily are made aware of our equilibrium. Or we may explain it thus:[1] if from every side A B, &c., we usually receive certain visually produced sensations with corresponding motor outpour, and we are suddenly deprived of those on one side A, then our outpour of energy tends to be limited to one side B, and to drive us over in the direction of A. This, opposed by the other senses, causes giddiness; but if we give attention only to the sight of the chasm, we actually fall. If we traverse a plank over a gulf, there is giddiness, but none if the plank be on the ground. Waltzing, stopping after waltzing, or looking at others doing so, equally cause it by the comparatively slow motion of the eye failing to keep up with the object, and then lapsing into irregularity. In sea-sickness there is disagreement between the eye, the touch, the muscular sense and the sense of equilibrium, or, Cyon says, between our perceptions at the time, and our standing representation of ideal space obtained through the canals. In the *Agoraphobia* of Westphal and others, the patients who usually guide themselves by the vertical lines of neighbouring things, break down in crossing open spaces. There is not actual giddiness, because, perhaps, no conflict between senses, only an absence of direction from one.

Equilibrium thus depends on the agreement of the sensations from both our own bodies, and from surrounding objects, and as

[1] Others explain this again by saying that we are unable to measure the depth of the chasm, and this lack of measurement causes the loss of equilibration.

soon as the sensations disagree, vertigo follows.[1] Revived mental states are a still more interesting cause, but I have no time to discuss them.

In conclusion, I have tried to examine some of the typical cases of vertigo, to inquire *whether it be a perversion of some normal function*, and we have seen that it can generally be referred to a consciousness of disproportioned attempts to maintain equilibrium, there having been in our cases either such attempts, or processes associated with them, or affecting the machinery for carrying them out.

This mechanism most probably includes the cerebellum, acted on by impressions, mental, visual, labyrinthine, tactile and muscular, obtained through various end organs. It has seemed needless to refer such a definite and elaborate sensation to changes in the brain-circulation only when there are definite organs able to produce vertigo upon stimulation; and without accepting as completely proved any theory of the canals, or Dr. Woakes's theory, there is strong reason to refer most of the excentric vertigo to processes acting through them on the cerebellum. In what single particular is any stomachal or toxic vertigo different from that of Ménière's disease confessedly acting through them?

At the same time I am aware of the danger of forgetting the plurality of causes, and of reducing the infinite variety of nature to the meagre uniformity of the human mind. The difficulties of getting evidence on a subjective symptom such as this are great; but so far as I can judge, there are two types; the subjective, where the person himself seems to move, produced by direct injuries to the cerebellum, epilepsy, &c.; and the objective, which includes aural stomachal varieties, and those caused by rotation, diseases of touch or sight, or affecting the circulation.

BIBLIOGRAPHICAL REFERENCES.

BROWN-SÉQUARD: *Cerebellum*, &c., in 'Lectures on Central Nervous System.'
HANDFIELD JONES: *Giddiness*, in 'Functional Nervous Disorders.'
HUGHLINGS-JACKSON: Articles in 'Lancet,' p. 727, 1874, and p. 675, 1877, and p. 525, 1880, and "*On Ocular Vertigo*," 'Ophth. Hosp. Rep.' v. viii., and 'Med. Times and Gazette,' p. 68, 1863.—"*On Auditory Vertigo*," 'BRAIN,' vol. ii. 1880, p. 29.
ROSS: *Vertigo* (i. 92, 408, 436), *Anæmia of Brain* (ii. 612), *Tumours in Cerebellum* (ii. 597), in 'Diseases of Nervous System.'
FOSTER: *Hearing* and *Muscular Sense*. 'Text-book of Physiology,' ninth ed.
CYON: Paper, 'Comptes Rendus,' Dec. 31, 1877, and 'Mind,' 1876.
PURKINJE: Article in 'Rust's Magazine,' 1827.

[1] As Wundt (Physiologie) remarks, so soon as the sensations which relate to space conditions are altered, vertigo (Schwindel) occurs, because all our volitions with regard to a composite action are strictly proportioned in each elemental part, though we placed before our consciousness only the attainment of a general aim.

CARPENTER: *Vertigo*, &c., in 'Mental Physiology.'
OLIVER: Article in 'Journ. Anat. and Physiol.' vol. xiii.
BROADBENT: Articles, 'Brit. Med. Journ.' p. 902, 1875, and April 1876.
ROBERTS: *Vertigo*, in 'Handbook of Medicine.'
JAMES: Article in American 'Journ. of Otology,' Oct. 1882.
ROOSA: *Ménière's Disease*, in 'Diseases of the Ear.'
RINGER: Articles on *Quinine, aconite, chloral, hot-water bags to the spine*, &c., in 'Handbook of Therapeutics.'
WOOD: *Quinine, aconite*, &c. in 'Text-book of Therapeutics.'
PRITCHARD: Article in 'Quarterly Journal of Microscopic Science,' Oct. 1876.
BRADBURY, J. B.: 'On Vertigo.'
GRAINGER STEWART: *Giddiness*, in 'Lectures,' 1884.—See 'BRAIN,' p. 137, 1884.
KNAPP and MOOS: Art. in 'Arch. of Ophthalm. and Otology,' vol. ii. No. 1.
NOTHNAGEL: *Anæmia of Brain* in Ziemssen's 'Encyclopædia,' vol. xii.
MERCIER: *Inco-ordination*. 'BRAIN,' xxi.
NIEMAYER: *Epilepsy*, 'Text-book of Pract. Medicine.'
ERB: *Vertigo* and *Ataxy*, in Ziemssen's 'Encyclopædia,' vol. xiii.
WOAKES: 'On Giddiness and Disease of the Ear,' and Arts. in 'Brit. Med. Journ.' Dec. 1882.
BASTIAN: *Cerebellum, Semicircular Canals*, &c., in 'Brain as an Organ of Mind.'
SPAMER: Art. in Pflüger's 'Archives,' vol. xxi.
BRISTOWE: *Vertigo, Ménière's Disease*, in 'Theory and Practice of Medicine.'
MOXON: *Influence of Nervous System on Circulation* (the Croonian Lectures), 'Brit. Med. Journ.' 1881.
MACBRIDE: *Auditory Vertigo*, 'Brit. Med. Journ.' 1882, and in 'Edinburgh Med. Journ.' 1880 and 1881, and 'Medical Times and Gazette,' pp. 96, 118, 1881.—'Guide to the Study of Ear-diseases.'
CHARCOT: *Vertigo*, &c. in 'The Diseases of Nervous System' (Sydenham Soc), series i. pp. 194, 212, 306; series ii. pp. 261, 265.
BAIN: *The Muscular Sense*, &c., in 'The Senses and Intellect.'
LEWES: 'The Physical Basis of Mind.'
HINTON: on *Diseases of the Ear*, in Holmes' 'System of Surgery,' 2nd ed.
DALBY: on *Diseases of the Ear*, in Holmes' 'System,' last ed., and 'On Diseases and Injuries of the Ear.'
GELLÉ: Art. in 'Gazette Méd. de Paris,' xxi. 1880.—*Labyrinthine Vertigo*, 'Archives de Neurologie' for 1882, &c. Abstract in 'BRAIN,' Vol. VI. p. 569.
FERRIER: *Cerebellum*, &c., in 'Functions of the Brain.'—Article in 'West Riding Asylum Reports,' vol. v.
MACKENZIE: *Vertigo*, in 'Quain's Dictionary.'
POLITZER: *Ménière's Disease*, in 'Diseases of the Ear.'
RAMSKILL: *Vertigo*, in Reynolds' 'System of Medicine.'
REYNOLDS: 'On Vertigo,' 1853.
TROUSSEAU: Arts. in 'British and For. Med. Chir. Review,' p. 51, 1864; 'Clinical Lectures,' vol. i. p. 55, vol. ii. p. 537.
WILKS: Arts in 'Med. Times and Gazette,' p. 83, 1869, and p. 422, 1879; and in 'Lectures on the Diseases of the Nervous System,' pp. 314, 339.
WUNDT: *Vertigo*, in 'Physiologische Psychologie,' Band I.
TOYNBEE: *Ménière's Disease*, in 'Diseases of the Ear.'
CRUM BROWN: Art. in 'Journ. of Anat. and Phys.' May, 1874, and in 'Nature,' 1874.
MACH: Art. in 'Sitzungsber. der K. Acad. der Wissenschaften,' 1873.
VULPIAN: 'Physiologie du Système Nerveux,' p. 601.
MÉNIÈRE: 'Bulletin de l'Académie,' xxvi. p. 24. 'Gazette Méd.' p. 83, 239, 547, vol. xvi. 1861.
ORMEROD: *Epilepsy in its relation to Ear-disease*, 'BRAIN,' Vol. VI. 1883, p. 20.
LIVEING: *Vertigo et passim*, 'On Sick Headache.'
BUZZARD: *Lectures on the Diseases of the Nervous System*; and Art. in Ranking's 'Abstract,' 1869, p. 29.
GASQUET: Art. in 'Practitioner,' p. 81, 1878.
ALLBUTT: Art. in 'St. George's Hospital Reports,' vol. viii.
LUCIANI: *The Functions of the Cerebellum*, in 'Brit. Med. Jour.,' Dec. 10, 1884.

Reviews and Notices of Books.

Sleep-Walking and Hypnotism. By D. Hack Tuke, M.D., LL.D., F.R.C.P. London, Churchill, 1884. 1 vol. 8vo. pp. 120.

Dr. Hack Tuke has done admirable service by associating the subject of sleep-walking with that of hypnotism and the so-called mesmeric state, for by so doing he has shown the common ground on which all these conditions depend, and has employed a method which affords a clue for unravelling the various phenomena exhibited under these different names. In all these peculiar manifestations of the human mind the essential feature is the partial or total loss of the ordinary volitional or guiding power, and the predominance of an instinctive or automatic form of action, in which there is often a complete subjugation of the faculties to the will of another person. At the present day the question awaiting solution refers to the actual state of the brain, and the nature of the mental processes which take place during these manifestations, and not to the mode in which they are brought about, for it would seem that from a variety of causes this automaton-like action may be displayed, and may therefore be regarded as one which is natural and common to all human beings. Probably every one under the rightly favouring circumstances might be thrown into this trance-like state, and therefore it is a phase which is absolutely necessary to be examined by those who make a study of the human mind. Formerly the automaton condition of which we speak was thought to be wholly abnormal or peculiar, and was to be induced only by some extraneous influence, such as the passage of the hand over the body of another person, through whom some special magnetic power was transmitted. This magnetic influence was supposed to pass from one person to another, and the object was said to be mesmerized; Mesmer being one of the earliest experimenters. When Braid found that any individual by gazing steadfastly at a bright object might fall into this trance-like state, it was obvious that no theory of animal magnetism was required to account for it, and now we are able to declare that even this simple process is not essential to produce it, for some persons may fall into this hypnotic condition without the use of any extraneous means whatever; and moreover, that somnambulism seems in every way identical with it. Dr. Tuke has therefore done well to insist on a study of the phenomena of

sleep-walking in connection with that of mesmerism or hypnotism, and to ask the assistance of others in making accurate observations of all cases of somnambulism. In the case of a girl, which Dr. Tuke reports, whether she fell into a state of somnambulism, or was thrown into the hypnotic state by another person, she displayed exactly the same phenomena under these different circumstances.

Probably modifications of the hypnotic state, where there are partial losses of consciousness, and yet various bodily actions continue, are common enough. Every one can relate in his own person of the performance of certain acts whilst he was altogether unconscious of them. In concussion of the brain, in drunkenness, and more especially in epilepsy, such temporary loss of consciousness is not uncommon. For example, the writer is acquainted with a medical gentleman who has had fits at long intervals. After one of these he found that he had lost his memory of acts which he had immediately before performed, and he in consequence requested his wife to accompany him on his rounds and take notes of all that he did. After visiting a patient and returning to his brougham, he made his wife write down the prescription from his dictation, and she would then direct him to the next place. The same process was repeated, until he had finished his rounds. When he arrived home he had no recollection whatever of the occurrences of the day. This forgetful state continued for three days, when his perfect consciousness returned. These circumstances were related by himself in the presence of his wife, who confirmed them. Moreover he never subsequently heard from his patients that any peculiarity had been observed in his manner during these three dream-like days. In these cases, as in those of somnambulism and hypnotism, the expression "loss of consciousness" is not altogether accurate, for the subjects of these states reciprocate to all that is said or done, and their senses are often very acute. A state of unconsciousness is one of coma, where a patient cannot be roused, and his senses are dead; whereas in the hypnotic or mesmeric condition the perceptive centres are still active, and the senses open to impressions, but the highest centre associated with clear mental consciousness appears to be dormant, and is not roused into activity by the ordinary stimulants. A condition somewhat similar is seen after concussion of the brain: for example, a man receives a violent blow and is rendered insensible; after a time he partially recovers and then acts like an automaton, he may walk about his room, dress himself, and perform other acts to which he is accustomed, but of all these he will not have the slightest recollection some days afterwards. There is thus apparently a lesser degree of the automaton state, that in which the person acts as if he were conscious, and yet is found to be soon afterwards quite oblivious of the scene in which he took part. For example, a man may in a drunken brawl resent insulting words, and be quite conscious of receiving a blow from another, and be able to retaliate; yet on the following morning he has no recollection of the fray, and the only proof to him of the fact is the loss of teeth, of which he was until then quite unaware.

In removing from the subject of trance or hypnotism the various causes which may induce it, we get rid of any discussion respecting mesmerism, animal magnetism, or other influence; we place it on a broader and simpler basis. We regard it as a normal condition, very common in a modified form, and only in extreme instances to be regarded as remarkable, as when seen in somnambulism, trance, or hystero-epilepsy. It is very probable that many of the death-like conditions into which animals may be thrown are of a like nature. At a lecture lately delivered by Mr. Langley at the Royal Institution, he had no difficulty in rendering a pigeon, a frog and an alligator perfectly motionless and perhaps unconscious. What we want to ascertain is what portions of the brain are in abeyance during this state, and which are active; also whether some portions are in a state of excessive activity, as for example those of the special senses. It is certain that the retina answers to impressions, for the somnambulist avoids obstacles, and can walk along the most dangerous paths, and yet the impression is not carried deeper into the sensorium, for some particular friend might be avoided as an obstacle, but not recognised. It is interesting to note modifications of this hypnotic or sleepless state as constantly occurring in ordinary life, for we thus remove it from the region of mystery and place it amongst the normal phenomena of the human mind. For example, the writer having been away from home for some time, returned to London, when on passing along a street he observed his father crossing at the end of it. The latter looked straight at him and walked on. Subsequently, when they met at home, the father said to the son, "I have seen you before to-day, but I do not know where." It is clear that his mind was preoccupied, and therefore received only the faintest impression of the image thrown on the retina, and this was not strong enough to arrest his progress. The various cases related by Dr. Tuke exhibit the condition of the senses in the somnambulistic state, in various degrees of activity, and thus although in some cases they are highly sensitive, in others they appear to be not in action, and as if the body was not so much influenced by them as by some powers within. For example, a hypnotic person whilst writing on a piece of paper would still continue to write on, even when a screen were placed before the eyes. This resembles what is sometimes seen in an epileptic, who will continue some mechanical movement after the fit has seized him, and he is quite unconscious.

The most remarkable phenomena which have to be studied in these cases are the acts which are due to suggestion. The subject of them appears to be like a pliant piece of machinery, ready to be acted upon or played upon by any extraneous force. He will apparently do anything he is bid, or believe anything which is told him Many of these phenomena are explicable on the ordinary theory of reflex action which accounts for a large part of our thoughts and sayings in the normal state; but there is one phenomenon which, if correctly observed, is most remarkable, and gives some sort of warrant for the old belief in animal magnetism. This is the influence which the operator has over the subject by merely

moving his hand when he stands behind the latter. It is credibly related that if the operator be behind him or her, and move his hand, the patient will approach or retreat as he beckons or warns off. If this is to be explained by the influence of the ordinary senses, it implies that the cutaneous surface of the patient is wrought up into a state of sensibility far beyond what is known in the normal condition, so that it becomes cognizant of vibrations of the air made at long distances. It was maintained by the late Dr. Beard, of America, that in hypnotism the perceptive centres and some other portions of the brain were in a state of hypersensibility, and he illustrated his meaning by pointing to a chandelier having a number of gas-burners. After comparing the full-lighted chandelier to the active brain, and the same when extinguished to the torpid brain, as in sleep, he took the case where one or two burners only were alight, and remarked the great height of the flames in consequence of their being supplied with a superabundance of gas whilst the others were not in use. This example he compared to the hypnotic state, where one or two of the senses were in a state of inordinate activity, whilst the other perceptive centres were dormant.

The whole subject is one of paramount interest in reference to the philosophy of the human mind, for it shows that consciousness is not an essential element in all our mental acts. It is now generally admitted that the higher centres of the brain may be in full operation without consciousness being called up, and to this the term "unconscious cerebration" is given. During sleep many instances might be related of the most complex mental operations being performed, and the subject of them being quite unconscious. He has only become aware of the recent activity of his brain upon waking up to the results in the morning. Although, under ordinary conditions, our mental state associated with consciousness directs our acts, we must admit that much must go on within us without consciousness or volition on our part. If this be the normal state, those of which we have been speaking, known as hypnotism or somnambulism, is a mere exaggeration of it; and if, as is generally admitted, reflex phenomena are everywhere apparent in mental as well as in physical states, we see that in hypnotism there is merely an extension or excess of a normal process. It can be easily understood how, the inhibitory actions of the will being removed, the brain can be more easily acted on through the senses.

Unless one has directed his attention to the fact, he is not aware of how large a part of our conversation in ordinary life is due to reflex action. The more a person can keep to the point of his argument, the greater his intellectual power; and, on the contrary, the being led away by new words or new thoughts is the very picture of weakness, and is little more than a representation of reflex action. All writers have discerned this, from Shakspeare to Dickens, in describing the speeches and conversation of illiterate persons. Albert Smith was in the habit of giving a sailor's yarn, in which one idea suggested another, and this a third, until the

story went on without any single idea running through it, and in which the starting-thought was totally lost. In a less degree, in ordinary table-talk, the subject started for conversation is often altogether forgotten; words or names let fall suggest some new thoughts, these when expressed suggest new ideas to others, until the company seem little better than automata. The more the subject is examined, the more intimately we shall find our thoughts and actions due to reflex action, so that we sometimes "answer a question without a thought," and for the time being are acting the part of the somnambulist. Dr. Tuke has done wisely by bringing together a number of allied phenomena, and showing how they are parts of the physiological law; how in various degrees, the guiding or inhibitory action of the will being removed, the lower functions of the brain can be set in operation through the senses.

Dr. Tuke's book naturally touches upon subjects of great practical importance, which are an outcome of these exceptional mental conditions. For example, the responsibility of the subject for acts done in the somnambulistic state, a question of the gravest importance, and one which had had to be discussed in tribunals of law. The following case told to the writer by a military friend might have had a very tragic result. When young, he had occasionally walked in his sleep, and whilst in camp in India, he one night found himself in the open country with nothing on but his nightshirt. He at once realised what had happened, looked about him to discover where he was, and hurriedly returned to his quarters. On entering the camp, the sentinel on duty presented arms after the usual fashion to an officer, and let him pass. It then became evident that the same formalities must have been gone through when he made his exit; he thought it prudent, however, to say nothing of his adventure, and therefore made no enquiries of the guard. There can be no doubt that if a private soldier had been seen walking out of barracks in a similar manner it might have fared very differently with him. We strongly recommend Dr. Tuke's book for perusal, for, although small, it contains facts of a most valuable and interesting kind, which must be thoroughly examined before a complete science of the human mind can be attempted.

S. Wilks.

A Text Book of Human Physiology. By Dr. L. Landois, Professor of Physiology in the University of Greifswald. Translated from the fourth German edition by William Stirling, M.D., Sc.D., Professor of Physiology in the University of Aberdeen. Vol. I. 8vo., pp. 514. London: Charles Griffin & Co.

Professor Landois' work on Physiology is particularly distinguished by its practical nature and by the constancy with which the Author brings the facts of Physiology into relationship with medicine. It

is a book written especially for medical students and medical practitioners, and the success with which the Author has adapted it to their wants is shown by the fact that it has already passed through four large editions in four years. Each section begins by giving a full description of the normal processes of life, and this is followed by a short account of their pathological variations, and a brief notice of the ways in which they are affected by various drugs. The work is thus calculated to direct the attention of the student towards a rational system of treatment, and to help the practitioner rightly to understand and treat the cases under his care. With this object in view, all the methods of investigation which may advantageously be used in practice are fully described, and more space is given to histology than is usual in text books of physiology. Professor Landois seems to have a special faculty for diagrammatic illustration, and his schematic drawings render the appreciation of histological and anatomical facts much easier. As illustrations of these we may mention his schematic drawings of the air-vesicles of the lung (Fig. 99), of the gastric mucous membrane (Fig. 136), and of a lobule of the liver (Fig. 141).

Professor Stirling has translated the work well, although here and there we notice a few imperfections. The most striking is his translation of the term "Selbst-steuerung," applied by Brücke to the action of the heart in producing ventricular dilatation as a consequence to ventricular contraction. Brücke supposed that during the ventricular systole the semi-lunar valves covered the opening of the coronary arteries, so that blood could not enter into them, but during ventricular relaxation the valves left the coronary openings free, so that the blood flowing from the tense aorta into the walls of the ventricle would tend to dilate it actively, and thus fill it more completely with blood for the succeeding systole. This action is like that of the valve which is attached to the cylinder of a steam-engine, and causes the movements of the piston to alternate. To this kind of gearing the term "Steuerung" is applied; but Dr. Sterling, instead of translating "Selbst-steuerung" as "valve-gearing," or "automatic regulation," or "automatic valve-regulation," or something of the kind, has translated it "Self-steering." No doubt the word employed by Brücke sometimes has the meaning given to it by the translator, but this does not seem to us to be at all appropriate to the action of the heart. The translation in general is, however, very good, and freer from German idioms than such translations usually are.

The work is, however, not a mere translation.—Dr. Stirling has made large and valuable additions to it. In places where the German edition begins abruptly, and seems to assume an amount of knowledge which the student may not possess, Dr. Stirling has supplied the necessary introduction; he has filled up any lacunæ that were present either in the histological descriptions or accounts of the functions of the various organs, and he has added descriptions of several methods of investigation omitted in the German work. The present volume contains the physiology of the blood, of the circulation, of digestion, of absorption, of animal heat, and

of tissue metabolism. Dr. Stirling has added 70 woodcuts to the 106 contained in the German edition—and has thus still further added to the value of Professor Landois' work, which itself is one of the best and most practical treatises on Physiology we have ever seen.

T. Lauder Brunton.

A Treatise on the Theory and Practice of Medicine.—By John Syer Bristowe, M.D., F.R.S., &c. London: Smith, Elder, & Co., 1884. 1 vol. 8vo., pp. 1240.

Lehrbuch der Speciellen Pathologie und Therapie der inneren Krankheiten. By Adolf Strümpell, M.D., Professor of Medicine in the University of Leipzig. Leipzig: Vogel, 1883-1884. 2 vols., 8vo.

Dr. Bristowe's treatise is so well known that it is unnecessary on our part to do more than welcome the fifth edition, and congratulate the author upon his well-deserved success. The part relating to nervous diseases occupies about 250 pages, and has from the beginning been a distinguishing feature of the book, which was the first of its kind in England to offer to the student a clear and reliable digest of recent neuropathology. Dr. Bristowe has completed his survey by adding a chapter on Insanity, including general paralysis, a subject of the highest importance for the general practitioner, as well as the student of the nervous system and its ailments, but which is still too often relegated to special treatises.

Professor Strümpell's textbook is a larger work than Dr. Bristowe's; but from the reception it has had in Germany at the hands of both students and practitioners, it bids fair to replace the old favourite volumes of Niemeyer. From the author's reputation as a neurologist, it was to be expected that the portion of the book relating to nervous diseases should be worked up with special care and success. Our anticipations have not been disappointed; and the first part of the second volume, which forms a distinct whole and contains 440 pages, gives us a most accurate and fairly full *résumé* of what is known on the subject it discusses. About 50 illustrations assist the reading of the text, which is written in a style, the lucidity of which many of the author's countrymen would do well to imitate.

We shall not attempt to criticise in detail the various chapters of the book, especially as we know that a second revised edition is now occupying the author. But we wish to draw the attention of the reader to the view taken by him of certain acute inflammatory processes. In a lecture published about a year ago,[1] Prof. Strümpell has given a clear account of the etiology of systematic

[1] 'Ueber die Ursachen der Erkrankungen des Nervensystems.' Leipzig: Vogel, 1884; pp. 22.

nervous diseases. He mentions and discusses four orders of causes: embryonic predisposition, over-use, psychical excitation, and toxic influences, among which "germs" play an important part. Acute poliomyelitis, acute multiple neuritis, and a form of acute hydrocephalus, are instances of nervous diseases of infectious nature, according to the author. Even those who are not prepared to go the full length with him will not fail to recognise the importance of the subject. As Professor Grasset says in his article on the relationships between hysteria and the tubercular diathesis,[1] our modern neuropathology has hitherto been absorbed in the study of lesions, and in the explanation of symptoms; of disease itself, the cause of those lesions, we know as yet very little. A true etiology is not only required for a complete knowledge, but is the condition of success in the treatment of nervous diseases. "Sublatâ causâ, tollitur effectus," should be the motto of our therapeutics, which are still in most cases painfully inadequate even to relieve the symptoms against which they are directed. But we need not despair, in presence of the rapid developments taken by neuropathology in the course of the last few years, and of which a most striking proof will be found in comparing the two Handbooks now before us with those in use during our own student-days.

A. DE W.

L'Homme et l'Intelligence. Fragments de Physiologie et de Psychologie. By Professor CH. RICHET, Agrégé à la Faculté de Médecine de Paris. Paris: Alcan, 1884. 1 vol. 8vo. pp. 570.

Des Suggestions Hypnotiques. By Dr. A. PITRES, Professor in the Faculty of Medicine at Bordeaux. Bordeaux: Feret, 1884. 1 vol. 8vo. pp. 64.

De la Suggestion dans l'état hypnotique et dans l'état de veille. By Dr. BERNHEIM, Professor in the Faculty of Medicine of Nancy. Paris: Doin, 1884.

THOUGH very unlike in title and external appearance, we may well group these three books together. Professor Richet assembles in one volume seven essays written at different times, but having a common aim—that of studying mental phenomena in connection with their physical embodiment and their cerebral substratum. The first subject, "Pain," and "The Causes of Disgust," are treated by the method characteristic of the physio-psychological analysis. In the second, "The Causes of Disgust," the author tries to reduce the phenomena of physical and moral disgust to a common law. He argues that in the midst of all the diversities of human tastes there runs a uniform thread; by following which up to its origin

[1] 'BRAIN,' 1884.

we reach the common principle at the root of all. This principle is that we are led to avoid hurtful, or even useless, things; disgust therefore may be defined as being primarily an instructive feeling, the result of hereditary experiences, of self-protection; but which, by association of ideas, is liable to almost infinite extension and fluctuation.

The Essay on the "Poisons of Intelligence," discussing the psychical effects of alcohol, chloroform, hashish, and opium, is full of interesting, if not always new, matter. Psychology must become experimental in order to become more exact, and cease to be a mere description of subjective phenomena, in order to become truly scientific. It must also be subjected to the comparative method in two directions; seeking to unravel the complex phenomena of our own minds, first by discovering the primordial constituents of conscious life in the lower organisms; second, by observing the disintegrating effects of disease. The superiority of the experimental method lies in the fact that the conditions of observation may be altered at will, though within necessarily narrow circuits, and that the observer may apply the method of introspection by using himself as the subject.

The chapter on intellectual "Poisons," and the next on "Artificial Somnambulism," contain many illustrations of the value of the experimental method; whilst the two Essays on Hystero-epilepsy ("Démoniaques d'autrefois et d'aujourdhui,") contain psychical studies of this now familiar disease. The last study, "The King of Animals," is of a somewhat different character, and treats of the relationship of man to nature, and of his rights over the lower animals; the cause of experimental physiology and pathology finds an eloquent advocate in Professor Richet, who with much reason points out that if the utilitarian argument that animals are not to be sacrificed for the sake of gratifying our curiosity had been enforced in the case of Galvani, we should be now deprived of all the boons which the discovery of dynamic electricity has conferred upon the human race. A copious appendix of additional matter, in the shape of notes to the several subjects discussed, closes the book.

The Essay on Somnambulism—which occupies 110 pages—contains many valuable facts and views; and should be read along with the pamphlets of Professors Bernheim and Pitres by all those interested in the study of the human mind. The depth of the somnambulic state varies considerably; three stages have been described, of which the first, or preliminary, period is called that of torpor; the second, of excitation; the third, of stupor; completeness of the hypnotisation is measured by the degree of automation to which the subject has been reduced. Experiments on animals may be of use to prove the reality of the phenomena, but cannot lead to the same results as those on man, who alone is susceptible of displaying the characteristic phenomena of suggestion.

These phenomena depend upon an alteration in the normal cerebral equilibrium, and dissect, as it were, the complex mani-

festations of psychical life; some components of which are abolished, whilst others are placed in relief, and may even be exaggerated. The inhibitory influence of the higher centres being in abeyance, the so-called spontaneous exercise of mind is abolished; there remains nothing but certain manifestations of memory, and association of ideas strictly limited to motives supplied by the operator; the subject becomes a cerebral automaton. In this somnambulic state, also, the mutual action and reaction of mind and body upon one another are displayed in a remarkable manner; not only are certain suggested trains of thought accompanied with appropriate actions, but postures expressive of feelings, passively impressed upon the limbs, call forth the corresponding trains of thought. Under the name of "Objectivation of types," Prof. Richet describes a state in which the subject, having lost the memory of his former self, acts and speaks exactly as another person whom he is made to believe to be.

A remarkable phenomenon is that whilst the subject may lose the sense of his identity, he does not necessarily lose the memory of past events, and yields a personal reaction, as it were, to external stimuli. The whole subject of memory with reference to the hypnotic condition is one of the deepest interest. As a rule, no reminiscence of what has been going on in that state is preserved after the waking up, though it may revive during the next hypnotisation. But very important facts have been brought to light recently, showing that in sensitive subjects suggestions made to them during their somnambulism may go on acting as motives to their actions in the waking state. They then feel a blind, uncontrollable impulse to do a certain thing, possibly shameful or criminal; they struggle against the impulse, and may overcome it if the action is sufficiently repugnant to their ordinary nature, but not otherwise. The influence of the suggestion may remain dormant for a considerable period; thus Prof. Bernheim relates a case where an order given during the hypnotic condition to be fulfilled two months afterwards, was fatally executed after that interval. But we cannot enter upon the details of these and the many other interesting observations related in the works before us; we hope to have said enough to stimulate the curiosity of the reader, and make him desirous of acquainting himself with the originals.

A. DE W.

The Medical Chronicle: a monthly record of the Progress of the Medical Sciences.—Edited by JAMES NIVEN, M.A., M.B., and W. J. SINCLAIR, M.A., M.D. Manchester: J. E. Cornish.

WE heartily congratulate our Manchester *confrères* upon the excellence of the opening numbers of their new periodical. They have pitched their standard very high, and for the sake of English medical literature we hope they may sustain the effort. As the title implies, the Journal is chiefly devoted to abstracts of recent

publications, which occupy about three-quarters of the entire number of 120 pages. These abstracts are carefully selected, conscientiously written, and form a solid body of readable matter, occasionally assuming the shape of short reviews or well-digested reports; and among them we may mention the admirable contributions of Dr. Ross on neurological subjects.

About thirty pages are devoted to original matter, consisting of short articles on points of practical interest. Let us note, *en passant*, that to a contribution by Dr. Railton on pseudo-hypertrophic paralysis, succeeds—an instance of *true* hypertrophy—a paper on the pulse, due to the imaginative exuberance of the ubiquitous Mortimer Granville. Otherwise the Journal does honour to the staff and their associates, among whom so much earnestness and ability is manifest. We wish them every success in a literary enterprise worthy of the rapidly rising school in the midst of which it has taken birth.

A. DE W.

Clinical Figures.—(Brain, etc., Series III.) Lebon and Co., 23, Southampton Buildings, Chancery Lane, London.

THIS third book of Diagrams of the Nervous System includes a horizontal section of the Brain through the internal capsule and basal ganglia (after Flechsig); a section through the crura cerebri at the level of the anterior pair of corpora quadrigemina (Schwalbe); one through the medulla oblongata through the middle of the olivary body (Schwalbe); and three transverse sections of the cord (after Flechsig) in the cervical, dorsal, and lumbar region respectively. They are very accurate; and, like their predecessors, will be found of great use, not only at the post-mortem table, but at the writing-desk also, for taking notes of published cases. Authors will be glad to know that Messrs. Lebon are prepared to furnish electro-plates of the figures, with lesions marked on them on the plan mentioned at page 280 of the present volume of "BRAIN."

A. DE W.

Abstracts of British and Foreign Journals.

A Case of Fatal Pemphigus-like Dermatitis, with Changes in the Nervous System.—By Dr. P. Mayer, of Strassburg. (Abstract from *Virchow's Arch.*, Part II. 1883.)—The patient was a man, æt. 65 years, and was admitted into the Strassburg hospital on November 28th, 1881. There was no neurotic family history, nor any evidence of previous disease in the patient, except that he had a double cataract.

The disease began three weeks previously with severe itching, followed by an eruption of small hard papules, which became vesicular and dried into crusts.

On admission, there was no disease of the viscera, but on the front of the chest was an eczematous-looking eruption, and on the neck some small itching papules. The eruption remained like an ordinary eczema for a week, and then pemphigus bullæ came on the trunk and limbs, with general disturbance, evening fever, frequent pulse, lithæmia, anorexia and sleeplessness, and pemphigus foliaceus gradually declared itself; large patches of cutis became discoloured, the epidermis was thrown off with very little fluid exudation underneath, and the aspect was that of a severe burn. Fresh eruption-patches came every day; but after a time bullæ ceased to form, diarrhœa set in, and he died on December 31st, seven weeks from the commencement of the eruption.

Post-mortem, there was nothing of importance in the viscera. There was a very little fluid in the dura mater spinalis, and the inner surface was pale; the anterior roots, especially in the dorsal regions, were rather thin, the cauda equina was strongly injected, the nerve fibres were rosy, the posterior roots flattened, and the cord was of normal consistency. In the neck the white substance of the posterior columns near the central canal was somewhat transparent, had a yellowish hue as far as the upper dorsal region; but here a greyish streak began on each side of the posterior longitudinal sulcus; in the posterior columns the grey substance was everywhere sunk in. In the lower dorsal region were transparent

patches; in the lateral columns, more marked still in the lumbar region. There were small hæmorrhages in the galea capitis—a recent hæmorrhage on the under surface of the dura mater on the right, small hæmorrhagic-spots on the left; the pia mater was œdematous; there was nothing particular in the brain. In the cord, one centimetre below the decussation, the posterior columns were greyish-yellow.

Histological Investigation.—Small pieces of skin were put into osmic acid, and large ones into alcohol and Müller's fluid.

Microscopical examination of the skin showed simply dermatitis, with considerable cell-infiltration and loss of epidermis. The subcutaneous nerves of the fore-arm, dorsum of foot and leg, showed many empty nerve-sheaths with numerous nuclei, and scattered myeline drops; and some nerve fibres were in the more recent stages of parenchymatous degeneration, with breaking up of the medulla and accumulation of granular protoplasm, proliferation of nuclei in Schwann's sheath in many fibres, and the empty sheaths had moniliform swellings filled with myeline balls, and the protoplasm rich in nuclei; here and there completely granular cells had already formed, which in varying number clothed the empty sheaths and very fine fibres with short links, and thin myeline coverings were scattered about. Many other fine skin-nerves from various parts showed signs of degeneration, and even some of the fibres of the sciatic, and larger branches of the brachial plexus. There was nothing in the muscles or sympathetic nerves, nor in the spinal ganglia or roots. In the cord—to the naked eye there were streaks along the longitudinal fissures in the whole cervical and dorsal regions, greyish-yellow discoloured places in the posterior root bundles in the upper cervical, the transparent spots in the lateral columns of the lower dorsal and lumbar regions. Microscopically—the interstitial tissue in these parts was thickened, the nerve-tubes were surrounded by a firm connective tissue, strongly stained with carmine, but poor in nuclei, and here and there bands of fibrous tissue usually along the blood-vessels; the vessels themselves had their lumen narrowed from fibroid thickening; thickened fibrous tissue also surrounded the nerve tubes singly, and in groups near the vessels; and in parts the nerve tubes were shrunken and reduced in size, and denuded of their medullary sheath, being only visible as small coloured dots.

There were no granular cells, but only scanty corpora amylacea. The sclerosis of the columns of Goll ran in the whole length of the cord, strongest towards the posterior commissure; in Burdach's

columns it was more in scattered patches about the middle between Goll's columns and posterior root zones; in the lower dorsal and upper dorsal regions there was an increase of the interstitial connective tissue in the lateral columns, though only in the neighbourhood of the posterior roots.

The anterior roots were quite healthy. In the grey substance there was only marked pigmentation of many ganglia cells, as is usual in elderly persons.

Remarks.—The changes in the skin were on the whole in favour of pemphigus, though there was more cell infiltration than usual, according to the view of some authorities, and there were too many nerve-fibres affected in the skin for the changes to be other than pathological, while they were proved not to be senile in the skin or in the cord, by comparison with the same parts in an older man.

Whilst the cord changes were undoubtedly older than the skin lesions, it is quite possible that it was only when the lesions reached a certain degree that they were of importance.

The connection between sclerosis of the posterior columns, irritation of the lateral columns and inflammation of the anterior horns, has been pointed out by many observers; but it is rare to find bullous dermatitis without previous nervous symptoms having been noticed when anatomical nerve changes were found post-mortem.

Schwimmer brings forward several cases with bullous eruption and cord lesions; his cases resembled ours, but were more severe, with the same parts, the columns of Goll, and posterior parts of the white substance affected, the process having evidently started there in the neuroglia and vascular scaffolding.

The duration of the skin disease negatived Babes's idea, that the cord lesion might be consecutive to the skin affections. Similar skin affections with cord lesions, are scattered through literature:—Jarisch's is a case in point, called by him Herpes Iris. In a woman, æt. 61, who had had a bullous eruption on the face, trunk and upper extremities for five years before admittance, and isolated bullæ here and there, and some capsules and vesicles, there were no spinal symptoms; but on her death, two months later, from pneumonia, &c., the cord was affected from the 3rd cervical to the 8th dorsal, in the anterior horns, and there was also a sclerosis of the central grey matter. Jarisch considered the whole process a neuritis in Stricker's sense. Quinquaud's cases, which developed into pityriasis rubra, also had

changes in the nervous system, in the shape of a diffuse myelitis with increase of neuroglia and connective tissue round the vessels, and also extensive parenchymatous degeneration of the skin nerves.

It thus appears that, as the result of pathogenetic irritation we may have papules and vesicles, or bullæ, or exfoliation, and although these changes are probably neurotic, coexistence is all that has been proved. It is to be remarked that the vascular changes in this case, consisting of obliteration of many of the skin vessels from endarteritis obliterans, and in the cord from a more diffuse fibrous change, was limited to those parts. It is quite possible that though this change was not recent, the nutritive changes led to sclerosis of the interstitial tissue of the cord, and when this was severe enough to compress the nerve fibres, the skin was damaged, its arteries blocked, and the process became so severe as to kill the patient. Further researches may show, whether the vascular changes in the skin and cord really possess the importance assumed by this hypothesis.

H. Radcliffe Crocker.

On Vasomotor and Secretory Troubles in Tabes.—Professor Pitres has reissued, in pamphlet form, a paper originally published in the 'Journal de Médecine de Bordeaux' (January 1884), wherein he records a series of symptoms which he observed in five cases of tabes dorsalis, and which, in the absence of other symptoms, he regards as of considerable value in forming a diagnosis. M. Strauss gave a description, in 1881, of the "Ecchymoses tabétiques," which may arise at any part of the body afflicted with lightning-pains, and the cases now recorded by M. Pitres agree with his in showing that these ecchymoses usually appear after attacks of pain, and wholly independent of any local traumatic influence. In themselves they are usually painless, are marked by much sanguinolent effusion, and occupy sites rather below the points of greatest severity of the lightning-pains, to which, indeed, they seem to bear some very definite relation. They rapidly disappear, and leave only a slight yellowish stain for a few days. In one of his cases which presented many of the more ordinary symptoms of tabes, the patient was subject to profuse sweating of the feet and lower part of the legs. This sweating was in fact the first symptom which, associated with painful tinglings of the feet, brought him under medical treatment. It was rapidly followed by impairment of vision and well-marked

ataxy, and after lasting six weeks suddenly disappeared, and gave place to violent lightning-pains in the same parts. He got worse during the next twelve months, and when seen by M. Pitres in June 1883, the skin of his feet and lower third of his legs was of a rose colour, which sometimes was found universally distributed, sometimes appeared only in patches or was occasionally absent, over these parts. The same area was covered continuously with small drops of sweat, which could be seen in course of formation, although the feet at the same time felt unduly cold. The nails of the great toes were crumpled and deformed. The fact that this local sweating appeared at times when the affected area presented no difference in colour from other parts which were perfectly dry, seems to show that the condition was wholly independent of any vascular disturbance, and to support the view that the vascular and secretory functions are not under precisely the same nerve control. Cases of this kind are by no means uncommon, and the symptom sweating bears a strong resemblance to those of increased saliva flow, vomiting and diarrhœa, which have been described by several writers as met with in the course of tabes dorsalis.

M. Pitres records an interesting example of *sialorrhœa* which occurred in crises, was ushered in by a strange sensation of burning on the top of the tongue, and lasted some two or three hours. The flow of saliva was enormous, had no obvious cause, and was unamenable to treatment. These cases are all brought together with the object of showing that certain symptoms of a rare and unusual kind may appear at the very beginning of the disease, before lightning-pains, gastric or other crises, or even ataxy have become established, and that their recognition is of great clinical importance. A case of the same character is also recorded in detail by MM. Raymond and Artaud ('Revue de Médecine,' No. 5, 1884, p. 414), where local sweating of the most remarkable nature occurred in the course of tabes. The authors give a short *résumé* of the various cases already published, by Nitzelandel, Eulenburg and Guttmann, Remak, Putnam and others, where symptoms were noted as being apparently due to disturbance of the great sympathetic. All of them have these common characteristics, sudden onset of the symptom, and great abundance of the secretion, whether it be from the salivary or sweat glands, or from the stomach or intestine. In their own case a soldier began at the age of 35 to have severe lightning-pains, which were followed in the next decade by many of the more usual symptoms,

occasionally by ecchymoses, by severe neuralgia in the distribution of the 5th nerve, by loss of teeth, squinting and impaired sight. He suffered when the authors saw him from severe lightning-pains in the legs, and each attack was accompanied with copious sweating from the affected limbs. Now and then, also, he has the *taches ecchymotiques*. Severe pains are also felt in the upper limbs, but more especially are they severe in the occipital and temporal regions, with giddiness and the sensation of a bar of iron piercing from one ear to the other. He has lost most of his teeth, and some of his nails are grievously altered. The most extraordinary feature in the case, however, is profuse sweating, limited to the right half of the head after each meal. About ten or fifteen minutes after eating he feels a sensation of pricking or warmth on the right side of the face, which forthwith is bedewed with limpid acid sweat which pours off in innumerable drops. There is little if any change to be noted in the colour of the right face at this time, its temperature is but very slightly higher than that of the left, and there is no alteration in the size of the pupil. The sweating occurs on the right side of the head and right shoulders, as well as on the face, but while it comes there only after lightning-pains, on the face it is after every meal. There is no defect or change of taste or smell, but the man believes there is increased flow of saliva on the right side while sweating lasts. The result of a subcutaneous injection of nitrate of pilocarpine was not different on the two sides. This symptom would appear to depend, say the authors, on some disturbance of function of the great sympathetic, in connection with disease of the spinal cord, and it is well known that degeneration is frequently found in those parts of the cord where M. Pierret, by recent researches, has placed the central origin of the sympathetic. The authors, however, say nothing about peripheral neuritis or degeneration, and it seems quite possible that many, if not all, of these strange symptoms may be due to peripheral disease of nerve-trunks, rather than to irritation of more central parts.

Féré and Others on the "Pied Tabétique."—We have before us no fewer than three papers on that rare form of arthropathy to which the name of "pied tabétique" has been given by MM. Charcot and Féré, in a description of the then known cases in the 'Arch. de Neurologie' (1883, t. vi. p. 305). Four cases were there recorded, and to them may now be added three more, possessing certain common features. M. Féré's more recent case, ('Revue de Médecine,' 1884, No. 6, p. 473), is that of a man aged

52, who came under observation in January of the present year, and who as long ago as 1872, after a severe fall, began to complain of violent pains having all the characteristics of the lightning-pains of tabes. At times also he had partial paralysis of the bladder with almost total impotence, and during the last two years occasional attacks of apparently causeless diarrhœa. There had, however, been little or no difficulty in walking, although when first seen there was unquestionable ataxy in gait, which with various other symptoms made the diagnosis certain. About two years previously his right foot had begun to swell without obvious cause, and changes in it had gradually supervened, so that when examined by M. Féré it was found thickened along the inner border, which had sunk lower than normal. This result is seen very strikingly in the impressions of the two feet, as shown in woodcuts accompanying the paper, for while that of the sound foot has a normal concavity on the inner side where the arch of the foot prevents contact with the ground, the whole sole of the right foot so touches the ground as to make one continuous line along its internal margin. The circumference of the right foot was found increased at the level of the cuneiform bones, and there was a well-marked projection of the dorsum, with a sensation of increase in the size of the first metatarsal and the internal cuneiform bones. The whole sole of the foot, however, seems somewhat shrunken. M. Féré enters into a consideration of the actual cause of the deformity, and looking into the construction of the arch of the foot points out that, from its form and build, destruction of the ligaments which retain the second and third cuneiform bones in place must lead to displacement upwards of one of them by the lateral pressure of the other two. He suggests that this displacement of a cuneiform bone is the real cause of the strange alteration in the shape of the foot.

The same journal (p. 486) also contains an account by Dr. Boyer of a case which fell under his care, and in which he had the good luck to be able to make post-mortem examination of the affected limb. The case is that of a man aged 45, in whom the symptoms began as long ago as 1870. A severe lumbago was then followed by violent lightning-pains, and in the course of a year or so by spontaneous dislocation of the right hip. After four or five years had elapsed, the man had extensive swelling of his right knee, which subsided and left the bones enlarged. About the same time both his feet were attacked in the same way, and were left enlarged and much deformed.

Not until 1879, however, did any ataxy become developed, but once begun, it rapidly increased. The patient died from "tuberculosis" in August 1883. Passing over other conditions, there were remarkable osseous changes in the right hip, in both knees and in the feet. The hip was completely dislocated, and the capsule of the joint was found converted into a thick osteo-cartilaginous shell, and there were numerous bony deposits—one especially large being just below the small trochanter, at the upper end of the femur, which with the ossified capsule formed a sort of new joint, the head and great trochanter having completely disappeared. The epiphyses of the tibiæ and femora had obviously undergone considerable atrophy, the spongy tissue thereof being soft, friable and vacuolated. The lesion of the feet was symmetrical, and consisted of enlargement of the astragalus, calcaneum and scaphoid, without anchylosis, and more notably of enlargement with anchylosis of the cuboid and three cuneiform, with their articulating metatarsal bones. The spongy tissue of all these bones was decidedly softened and friable. Yet another case of a like nature is to be found recorded in the same journal for May (p. 418), by MM. Gaucher and Duflocq, of "arthropathic tibio-péronéo-tarsienne gauche tabétique." At the age of 25 a soldier had a severe attack of cholera, and from that time onwards his disposition completely changed, and he became subject to attacks of great nervous irritability, and shortly afterwards, in 1850, to lightning-pains in the legs. About the same time, also, ataxy began, and, along with the pains, this continued in varying degree until 1869. In this year these symptoms became more prominent, and to them were now added anæsthesia of the soles, occasional diplopia, and loss of acuity of vision. During the siege of Paris in 1870-71 he rapidly got worse, became subject to hysteriform attacks, and occasionally had œdema of the lower part of the legs. During one of these complex seizures in 1881, the whole left leg became œdematous, but the swelling subsided after a few days' rest from all parts except the ankle and foot. Under prolonged treatment this indolent swelling of the soft parts almost entirely disappeared, leaving, however, very obvious bony enlargement of the lower part of the tibia. Again in the same year he had another attack of severe pain, followed by a like œdema, by a steady increase of the other symptoms, with incontinence now of both urine and fæces, and ataxy of the upper limbs. Leaving aside a minute description of the many symptoms present, the lesion of the ankle is thus

described when seen in 1883. Tarsal and metatarsal bones are unaltered, with the exception of some enlargement of the head of the first metatarsal. The anterior part of the internal malleolus is much enlarged, and presents a uniform smooth swelling, which gradually fades away into the shaft of the tibia, which thus appears to have a well-marked curve inwards. The most noteworthy change, however, is in the fibula, which at its lower fourth begins to be enlarged, and increases in size downwards, so as to present a large osteophytic tumour on its anterior part, which is separated from the external malleolus by a depression, which thus gives it the appearance of being a second external malleolus. These cases all present certain common features. In all, lightning-pains were prominent; in none was ataxy a pronounced early symptom, nor were there true gastric crises. In each the bony changes were preceded by severe attacks of pain, associated with sudden œdema of the parts, and these changes seem in all of them to have consisted of atrophy here, and of the growth of exostoses there. In all, the changes themselves and manipulation of the affected joints were almost painless. The cases are a valuable addition to those which have been already published, and they go to show that the small joints and bones of the foot and ankle, though less often attacked, are liable to the same sort of arthropathic affections in the course of tabes dorsalis as are the larger joints of the body. The whole series of cases, here given in briefest abstract, forms an important contribution to the study of this marvellous disease, which seems daily to widen the domain of its symptoms, and which may come under observation when wholly unexpected, and in the most deceptive guise. It is not without interest to note, that in four of the nine cases recorded in these various papers there is a clear history of syphilis, and that in none of them is any special mention made of rheumatism, either in the patients themselves or in their families. To the existence of this latter disease in those afflicted with arthropathies it would be well if future observers were to direct very careful attention, for the special character of these so-called Charcot's joint diseases is, as it is well known, doubted by very competent observers, and this point not less than others has yet to be cleared up with reference to the ætiology and pathology of tabes dorsalis.

Dubler on Neuritis in Herpes Zoster.—In an abstract of a previous contribution of MM. Pitres and Vaillard on "Non-traumatic Peripheral Neuritis" (see 'Brain,' Vol. VI. p. 555),

reference was made to a case of theirs, where inflammatory degeneration of two intercostal nerves was regarded as the cause of attacks of Herpes, and to their belief that microscopical examination had been too exclusively directed hitherto to central structures rather than to the nerve-trunks themselves.

Dr. A. Dubler now brings forward (Virchow's 'Archiv,' May 1884, p. 185) two cases of great interest, where by very careful examination the presence of peripheral neuritis was as clearly established as was the absence of central disease. In one, the inflammation of the intercostal nerves was clearly due to their being involved in some periosteal swellings of the ribs, from which points of irritation the degenerative changes had spread and involved their very finest endings both in muscle and skin. The cause of the neuritis was not so obvious in the other case, but the peripheral changes were not less pronounced. In this second case it is noteworthy that the spinal ganglion of one of the affected intercostals—the 9th—showed degenerative changes, but only in that part of it to which the degenerated fibres of the trunk itself could be traced, and as no such changes were to be found in the ganglia of the other diseased nerves, it seems probable that the neuritis may have begun as spontaneously in the ganglion as in the trunk, and that there was really no connection between the two. Plates are given of the microscopical appearances, which are seen to be of the same nature as have been described by Pitres and Vaillard. M. Dubler's paper is worthy of careful perusal, for in addition to his own cases, recorded at great length, he refers to numerous others which have been published, and he shows how really inadequate were the examinations made in them, and how necessary it is to subject the nerve-trunks themselves, throughout their whole lengths, to microscopy. It is indeed somewhat remarkable that the discovery of these peripheral changes is of comparatively recent date, for the suspicion has long existed that some change in the nerve-trunks themselves must underlie that form of Herpes which accurately follows the line of nerve-distribution. As long ago as 1869, Broadbent wrote in a paper on "Speculations concerning Herpes Zoster," that the "provisional hypothesis" at which he had arrived as the proximate cause of Herpes Zoster, was that there was "some undefined condition of a nerve-trunk, whether inflammatory or anæmic or other he was not prepared to say, or how it was induced, and that the particular part of the nerve affected was certainly not the root."

Schulz on the Importance of Tendon-reflex in the Examination of Suspected Simulation of Spinal-cord Diseases, (*Deutsches Arch. für Klin. Med.* Feb. 1883, p. 455.)—After giving a careful *résumé* of the different views which are held on the physiological import of the so-called tendon-reflexes, Schulz comes to the conclusion, from the clinical observation of numerous cases, that simulation of ankle-clonus or of the absence of patellar tendon reflex (knee-jerk) is a thing absolutely impossible. Two striking objective phenomena can thus, regardless of the patient's will, be always investigated in cases of suspected simulation of paralysis; and Schulz points out the value of this observation in cases of alleged injury from railway accidents. The instances have not been few in this country where the presence or absence of these phenomena has afforded invaluable aid in diagnosis when no objective sign could be discovered amongst overwhelming numbers of subjective symptoms, and Schulz tells how in Germany also it is especially after railway collisions that simulation of paralytic maladies is most likely to be found. Railway and post-office servants have learnt, he says, from one another the complex symptoms of the *railway spine*, and it is therefore most important for the surgeon to be armed with some means of diagnosis which is practically conclusive. Hardly sufficient stress is laid, perhaps, on the fact that both ankle-clonus and exaggerated knee-jerk may be sometimes met with in paralytic conditions not dependent on organic disease of the spinal cord or in certain functional states of the nervous system, but there is every ground for believing that true ankle-clonus and abolition of knee-jerk are absolutely beyond the active will of the patient. There yet remains a small percentage of cases, so small as scarcely to lessen the value of the phenomenon—where the knee-jerk is normally absent. Berger, for example, found it absent in 22 out of 1409 presumably healthy men, and in 694 school-children of both sexes examined by Block it was wanting in 5, of whom 4 certainly belonged to neurotic families, one of them, moreover, having curvature of the spine, and only one being absolutely sound. The difficulty of eliciting the knee-jerk is, moreover, great in short thick legs, as has been pointed out by Westphal; and perhaps we are as yet ignorant of some possible variations of the so-called reflexes within the limits of perfect health.

HERBERT W. PAGE.

Remak on Hypoglossus Spasms. (*Berl. Klin. Wochenschr.*

1883, No. 34.)—A man, 33 years old, suddenly felt numbness in the left half of the tip of the tongue, taste, feeling and movement being unaffected at first; the numbness spread in two weeks to the middle of tongue, the inner side of left half of lower lip and corresponding gum, and at the same time he had painless spasms of tongue, affecting him in speaking and eating. Slight paralysis of left facial nerve, as shown by lowering of left side of mouth, was present; the spasm of the tongue consisted in a flattening and narrowing of the upper surface, and rapid protrusion, forty times a minute, while corresponding rhythmical contractions of the muscles of tongue between the lower jaw and the hyoid bone occurred, and affecting especially the genio-hyoid muscles. Movements were increased when the tongue was protruded. Otherwise voluntary movements free in all directions. Sensibility normal; teeth healthy. Taste and sensibility of all kinds normal. Electric excitability normal; by tetanizing left side of tongue the spasms ceased. Speech not altered, except thick and slow, and talking did not increase the movements. Has often bitten his tongue. Movements increased by eating and not by drinking. Velum palati normal.

After some days he had severe clonic and tonic convulsions of the left side of the face, especially the buccinator, zygomatici, triangularis and quadratus menti, digastricus and platysma myoides muscles, while the upper branches of facial nerve were not affected; the patient was prevented from speaking or eating for one minute while this attack lasted. After these attacks the movements of the tongue were lessened, but the paresis of left side of face was rather more. Subsequently several convulsions occurred five or six times a day on eating and also spontaneously, while the original movements of the tongue stopped altogether. Remak quotes Romberg's opinion, that hypoglossus spasm associated with mastication is the rarest form of the disease.

He refers to cases of hypoglossus spasm produced by attempts to articulate. Also to one of Weir Mitchell's, with painful spasm of the tongue, spreading to the facial nerve, and cured by the extraction of a tooth.

From absence of pain or any peripheral irritation, a reflex cause cannot be substantiated. He considers that the lesion might be in the hypoglossus nucleus, and also in the left facial nucleus contiguous to it; or in the right ascending frontal convolution of brain, although there is no aphasia and no loss of sensation. Treatment such as by galvanic current and nerve-stretching would be

influenced by a correct regional diagnosis and by determining whether the disease is functional or organic.

The attacks diminished, and were finally cured in eight days by treatment with iodide of potassium, bromide of potassium, and daily stabile galvanic treatment with the anode pole to the infra-maxillary region and left side of neck and throat.

Rumpf on Influence of Narcotics on the tactile Sensibility of the Skin. (*Vortrag 2ter Med. Congr. Wiesbaden*, April 1883.)—He employed hypodermic injections of hydrochlorate of morphia, from $\frac{1}{4}$ to $\frac{1}{6}$ of a grain, and in 6 to 7 minutes a diminished cutaneous sensibility over the whole body was noticed which reached its maximum in an hour; the sensibility was tested by an æsthesiometer. He agrees with Jolly—and so differs from Eulenburg—that sensibility is not more diminished at the point of injection, than over the rest of the body; but he thinks that under pathological conditions the local effect might be stronger. Chloral hydrate, given by the mouth, produced less effect than was expected, and after a dose of 30 grains, sufficient to produce drowsiness, only a very slight diminution of sensibility was obtained. Alcohol by the mouth in doses of about $1\frac{1}{2}$ ounces diminished the sensibility, particularly in the lower limbs. Bromide of potassium in drachm-doses gave the most decided diminution of sensibility. Extract of hyoscyamus had, contrary to expectation, no particular effect. Cannabinum tannicum, the glucoside of Indian hemp, in doses of 4 grains by the mouth, caused considerable lessening of sensibility, and also produced calm sleep. Caffein, $\frac{4}{5}$ to $1\frac{3}{4}$ grain in an ounce of coffee had a very strong influence in increasing the sensibility of the skin, but the effect soon passed off. Morphia had the most effect, and bromide of potassium next.

Kast on Ataxic Movements in disease of the Spinal-Cord (*Nat. Forsch. Gesells. Freiburg*).—He discusses the theory of co-ordination, mentioning that there exists a hypothetical system, which receives motor impulses from the cortical centres, and directs them to the various groups of muscles; and if these motor impulses are deficient, owing to a lesion of the co-ordinating systems, or to some hindrance in the peripheral tracts, faulty combination is the result, and hence ataxia. He asks whether correct co-ordination is due to centripetal sensory impressions, or to purely motor impulses. Ataxia occurs in tabes, Friedreich's disease, disseminated sclerosis, and ataxia.

He quotes cases of Kahler and Pick, and Vulpian, with multiple tubercles of cord in the case of former, and spinal compression in that of the latter author, associated with ataxic movements. He gives in extenso the case of a man who had fracture of the dorsal vertebræ from a fall, extravasation in the dura mater, myelitis of the dorsal cord from compression, complete motor paraplegia, slight loss of sensation, gradual motor recovery in eight weeks, but with typical ataxia of legs, ending in complete recovery. When the ataxia was developed he had full motor power in the legs. In the right leg ataxia and loss of sensibility to touch and pain were more marked than in the left. Ataxia was not much increased on closing the eyes. Both knee-jerks readily obtained; no ankle clonus; plantar reflexes active; sphincters not affected. Left leg recovered before the right.

In another case of acute transverse myelitis of the dorsal cord, with complete motor and almost complete sensory paralysis of both legs to touch, temperature and pain, incontinence of urine and fæces, there was decided ataxia with return of motor power. Complete recovery in eighteen months. In this case the plantar reflex was active, cremasteric absent; knee-jerk well obtained, left more than right; ankle clonus on left, not on right. When he had a complete return of motor power the ataxia persisted, and was more marked in the right than the left leg.

Kast remarks that, in these two cases, a stage intervened between the complete motor paralysis and return of muscular power, in which, with almost complete motor power, there were considerable defects of co-ordination and ataxia. In these cases he thinks that either the extensive myelitic process involving the whole transverse section of the cord, had disappeared from all parts, leaving its effect most in the posterior columns containing the co-ordinating tracts; or that here the lesion was from the beginning most intense; or that for some reason, lying outside this tract, its recovery or that of its collateral fibres had been unduly retarded. He draws attention to the laxed condition of the muscles and joints, which was very marked in this patient, and which has been considered an explanation of the ataxia in these cases.

He quotes Tschirjew, who considers that the tracts for the preservation of muscle tonus are identical with those for the conduction of tendon reflexes; and the failing of the muscle tonus of the antagonizing muscles, produced by the interruption of the centripetal nerve tracts, causes the excessive and irregular movement of the limbs; and this disturbance of movement produced by the

lesion of these peripheral regulating apparatus, he calls peripheral ataxia. He also thinks that the absence of the tendon reflexes is caused by a slighter lesion of nerve fibres, than are the loss of muscle tone, and the ataxic movements of co-ordination, due to the tonus of antagonizing muscles; and thus tries to explain the incongruity of the presence of the tendon reflexes with the disturbance of co-ordination. Westphal and Senator oppose this theory; and Kast thinks that his cases prove, that absence of tendon reflexes is not necessary for ataxia.

Kast on Reflex Saltatory Spasm. (*Neurol. Centrbl.* 1883, No. 14.)—Patient, a man 39 years old, with no neurotic family history, complained of convulsive "drawing" of both legs from the calf upwards. Onset sudden. One morning he could not stand still on his feet, as soon as they touched the ground the heels lifted up and caused him to stumble and fall. There was some increase of patellar reflex; he got better after being in bed a month, and resumed work. Subsequently, when very tired from walking, he suddenly had cramp in the calves, severe shaking of both legs, and fell down, and the shaking occurred whenever he tried to walk. After seven weeks in hospital, with warm baths and constant current, he got better, but the same symptoms came on again when he was tired. When put on to his legs, the muscles contract rhythmically and his heels are raised off the ground, the shaking extends from the calf to thighs and buttocks; he staggers and falls. Muscular power intact. No tremor on voluntary movement when lying down, no inco-ordination, no rigidity. Sensibility normal. Muscle sense and electric state normal.

Patellar tendon reflexes excessive on both sides. Ankle clonus present, but sometimes prevented by a slight tetanic rigidity of the calf-muscles. Skin reflexes increased, plantar abdominal and cremasteric, in both lower limbs increased *tendon reflexes.* He got better with a generous diet, and was able to walk in ten days; but clonus of the calf-muscles could be produced when he was made to stand on his toes; tendon and skin reflexes gradually diminished till ankle clonus disappeared. Kast states that only nine cases have been noticed. He quotes Erb in support of the theory, that this disease is probably due to increased reflex irritability and especially of the tendon reflexes, and is rather a symptom of different diseases than a distinct form of spasm. Refers to a case of Petrina's, where the patellar tendon reflex was very weak, and later on was

not obtained, and ankle clonus was absent throughout; but this case was probably an acute disseminated cerebro-spinal disease. In Kast's case the spasms could sometimes be stopped by compressing the muscles or by cutaneous electric irritation, but not by voluntary impulses.

The term of reflex saltatory spasm was first given to these cases by Bamberger. Kast thinks that the saltatory spasm can occur as part of an acute disturbance of co-ordination, and can exist when there is extensive diminution or even absence of the skin and, tendon reflexes.

C. E. Beevor, M D.

Associated Systematic Sclerosis of the Columns of the Cord.—In the *Archives of Neurology* (January, 1884) Drs. Ballet and Marie report a case of sclerosis of combined systems of the cord which presented clinical and pathological aspects of much interest. The patient, four years prior to admission into Salpêtrière, suffered from amblyopia, and became completely amaurotic within a year, with the accompaniment of severe headaches, compared to the pressure of an iron band around his head: there was no vomiting. Three years subsequent to this, "lightning pains" began in the limbs, and cutaneous hyperæsthesia of the extremities rendered the least touch painful. Close questioning, however, elicits the fact that such pains might have occurred previous to the development of ocular symptoms. At this period (1881) the arm became first enfeebled, and three months later there was loss of muscular power in the leg.

Upon admission in February, 1882, he was bedridden from paralysis of the limbs; he was also completely blind, not distinguishing light from darkness. The ophthalmoscope revealed grey atrophy of the papilla. He complained of transient buzzing noise in the ears, with vertigo.

Motility.—There is advanced paralysis of upper and lower limbs, cannot walk or even stand erect; executes feeble movements, but with much effort while recumbent. Arms are slightly less affected than the legs. The paralysed limbs are rigidly contracted and the seat of cramps: deep reflexes notably exaggerated: muscles wasted: no paralysis of bladder or bowel.

Sensibility.—Transient, often recurring pains of burning character affect the legs, and are accompanied by numbness of feet; all the limbs reveal painful hyperæsthesia of the skin, except left leg, which is partially anæsthetic. Later on in the year, lancinat-

ing pains affect the chest, and are often localised in one spot in an intercostal space. Frequent syncopal attacks occurred during this year; and there is now loss of power over sphincters of bladder and bowel. For five days preceding his death he suffered from sharp pyrexia, ranging betwixt morning and evening from 102° to 103° Fah.: an eschar forms on sacrum: skin becomes citron-yellow, and the conjunctivæ of icteric tinge.

Autopsy.—Brain shows no disease beyond grey atrophy of optic nerves, chiasma and tracts, with greyish degeneration of the outer geniculate bodies.

Spinal cord, upon microscopic examination, shows a sclerosis involving both posterior and lateral columns: in the greater part of the lumbar cord these columns have escaped, but higher up nearly their whole thickness is invaded. It is only in the lowest cervical and highest dorsal region that the columns of Goll are completely altered, and the whole thickness of the posterior columns affected by disease: higher still, the columns of Burdach are untouched, save close to the pia mater; but the columns of Goll are utterly degenerated as far as the medulla oblongata.

There is sclerosic change of the lateral columns at all heights as far as the pyramidal decussation; forming a small wedge-shaped tract in the lower lumbar, it rapidly enlarges in the upper lumbar region, and from this point up to the decussation it involves nearly the whole depth of lateral columns, including the direct cerebellar and crossed pyramidal tracts. No change had occurred in the cells of the cornua.

Under strong objectives the following features are revealed in the diseased tracts:—

(*a*) Fresh preparations show an absence of granular and fatty bodies. (*b*) Sclerosis is not of the same degree at different sites: *it is denser next the pia*, and less dense in the posterior than the lateral columns. (*c*) Amyloid bodies are found at margin of diseased tracts. (*d*) Proliferation of neuroglia nuclei. (*e*) Numerous stellate cells, especially near peripheral parts in the lateral columns. (*f*) Large size and number of vessels, with walls and sheaths infiltrated with leucocytes. (*g*) Where the nerve-tissue is not completely destroyed, the morbid change resembles that of acute and subacute forms of myelitis, viz. "distended nerve-sheaths and varicose axes." (*h*) Anterior and posterior nerve-roots were normal. The optic nerve showed complete absence of nerve-fibres.

Our authors, upon reviewing these data, argue that this case is

not one of systematic myelitis, in the usual acceptation of that term. They emphasise the strange predilection shown by the morbid process for the dorsal region of the cord, and the integrity of radicular zones at lower lumbar realms — for tabes by preference attacks the lumbar cord. Then, as regards the lateral columns, they urge that authors frequently use as synonymous the terms "lateral" and "pyramidal" columns, instead of clearly distinguishing betwixt the centrifugal and centripetal tracts of this complex system. They insist upon the advance of sclerosis from the meningeal border of the cord inwards, through the cerebellar to the crossed pyramidal systems—breaking through physiological barriers and diffusing itself irregularly with an independence of behaviour which recalls the progress of multilocular sclerosis: it is *not*, therefore, a systematic but a diffuse sclerosis.

It is difficult to conceive a genuine systematic sclerosis of the lateral columns which should absolutely respect the integrity of the columns of Turck and anterior horns: yet M. Jubineau records three observations of such a character. It is of interest to note the atrophy of optic nerves which so frequently accompanies ordinary tabes, associated with a lesion of the posterior columns, which in this case appears developed by a process wholly differing from that of ordinary locomotor ataxy. The authors then summarise what appears to them to be various types of system-affections due to sclerosis of the posterior and lateral columns of the cord: these are as follows:—

1. Diffuse sclerosis, as illustrated by the case under consideration.
2. Classical ascending sclerosis, involving the columns of Burdach, Goll, and direct cerebellar tract, in virtue of their conductibility and physiological functions.
3. Combined case of primary systematic sclerosis, with a consecutive diffuse myelitis.
4. Systematic sclerosis, consecutive to diffuse interstitial myelitis.
5. Combined ascending and descending systematic sclerosis.

The widely dilated vessels surrounded by leucocytes is insisted upon as suggestive of a perivascular origin for the changes seen: as in the liver, so in the cord, the forms of sclerosis must be various. They suggest "peritubular" and "perivascular" as appropriate terms, since the terms specifying the sclerosis should indicate its point of departure; of course every perivascular sclerosis eventually involves the medullated tubuli and becomes peritubular. In connection with this statement, it may be remarked that a case

of tabes in conjunction with ophthalmoplegia externa was published by Dr. Buzzard in the April number of 'BRAIN' for 1882, in which similar notable lesions of the blood-vessels of the cord were described, and a second case by him in the January number of 'BRAIN' for last year, revealed such changes as induced him to regard the affection as primarily a peri-arteritis in origin. Prof. Adamkiewicz is regarded by Drs. Ballet and Marie as in error when he refers the classic form of locomotor ataxia to lesions of a perivascular connective change, rather than a peri-radicular implication, and they incline to support Charcot's and Pierret's views upon this point. In the case under consideration, a specific origin is claimed, there being a history of syphilis, and all similar cases are referred to probably some intravascular toxic or infective agency.

The authors next refer to the antagonistic nature of symptoms which may be supposed to arise in the combined systematic forms, viz.:—the acute pains, hyperæsthesia or anæsthesia of a posterior sclerosis, with its abolished reflexes and flaccidity of limbs; and the rigid contractures and exaggerated reflexes of a lateral sclerosis. In a case quoted by M. Babesiu, there were at first feebleness and heaviness of the legs: contractures, followed by pains in limbs and genitals: next, atrophy of disc towards the end, plantar anæsthesia, loss of muscular sense, paralysis of bladder, tendon-reflexes slightly exaggerated. In a case given by M. Raymond, the predominant symptoms were those of lateral sclerosis, although both lateral and posterior columns were affected. In the three cases, therefore, of Raymond, Babesiu, and that by Ballet and Marie, the lateral column maintained its rights to assert its implication by contractures and exalted deep reflexes, despite the grave lesions in the posterior columns. We note here, however, that in Babesiu's case the tendon-reflexes were but "*slightly exalted*," and it appears more natural to suppose the various degrees of modified reflexes would occur in these cases of combined systematic sclerosis sometimes more, sometimes less exaggerated, or more or less diminished, according to the predominance of the one or the other lesion. Dr. Buzzard, in his 'Lectures on Diseases of the Nervous System,' has specially drawn attention to the question propounded by Ballet and Marie. Our author's explanation of the phenomena is at all events ingenious: the columns of Burdach are supposed to be free from lesion in the upper cervical and greater part of lumbar regions; hence the reflex arcs are free for both extremities. The explanation is therefore one of *localisation*. So

for lesions predominating in the posterior columns, the advantage being with the degree of lesion, so to speak, if the whole height of the cord be affected there will be abolition of reflexes, and the limbs flabby or but slightly contracted; if, on the contrary, a large part of the posterior columns be intact, the sclerosed lateral columns maintain their rights to their usual mode of symptomatic expression. This is nearly Westphal's opinion: "When there exists an associated affection of the posterior and lateral columns, neither muscular rigidity nor contraction supervene if the lesion of the posterior columns extends as far as the lumbar enlargement, and if the radicular zone of these parts be degenerated."

Hemiplegia occurring during the course of Diabetes. (Drs. RAYMOND and ARTAUD: *L'Encéphale*, No. 3, 1883.)—It is generally admitted, since the researches of Claude Bernard, that certain lesions of the cerebro-spinal axis may induce glycosuria, whilst the latter affection in its turn may react on the nervous system, producing very varied symptoms of centric disturbance. Equally well is it established that amongst the most important accompaniments of diabetes are the initial monoplegias characterised by their transient, mobile nature, their incompleteness and tendency to affect individual musculatures, and the later hemiplegic seizures of more persistent type, often preceded by intellectual troubles like those of cerebral softening, and usually due to vascular lesion. The patient whose case is detailed was a female aged 68, with nothing peculiar in her former history until three years prior to admission, when very intense irritation of the genitals, with frequent copious micturition, commenced, accompanied by intense thirst.

Twelve months later, dimness of vision affected first the right and then the left eye. Upon admission, the lenses of both eyes were cataractous and patient nearly blind; there was no longer intense thirst; appetite and digestion were normal; there was obstinate constipation. Physical examination reveals very little wrong with chest or abdomen, but the urine is found to contain on an average 15 grammes of sugar per litre, and heat and nitric acid show much albumen. For a period of nine months this condition is maintained, but also supplemented by severe lancinating pains at the right occiput and progressive mental enfeeblement. The daily amount of urine passed, from 1500 to 2000 grammes, contained besides the usual amount of sugar about 1½ grammes of albumen per litre. The left arm is now affected by a progressive paralysis, which

slowly invades the leg also on this side; there is no facial or ocular paralysis.

The left arm is not completely paralysed; the grasp is, however, very feeble; sensibility to pain is somewhat diminished; reflexes and electric-contractility are normal. The condition of the leg is similar, except that the deep reflexes were exaggerated. The autopsy revealed a focus of softening in the right hemisphere, limited in front by the ascending parietal, below by the first temporo-sphenoidal, above by the postero-parietal and behind by the annectent gyri; the membranes adhered to the diseased part; no other portion of the hemispheres, pons and medulla or cord, presented any coarse change. Microscopic examination of the bulb and cord shows none of the changes described by Dickinson, and no secondary degenerations of crossed or direct pyramidal tracts. The organs, however, generally (liver, heart, kidneys) presented a notable degree of sclerosis spreading along the peri-vascular tracts. Strangely enough, the cut illustrating the article exhibits the lesion upon the left hemisphere.

Raynaud on a case of Cerebral Localisation.—Dr. Raynaud in the same number of *L'Encéphale* details a case bearing upon cerebral localisation. The patient, when admitted, suffered from right hemiplegia with incomplete aphasia (replies usually by gestures, sometimes by inarticulate sounds, interspersed with distinctly uttered words, such as "non," "coquin," &c.).

Three weeks later, a second apoplectic seizure with deep coma occurs, from which she recovers gradually, retaining pantomime and the former expressional power; but a few months later on she is found to have *entirely lost* the faculty of articulate language, and a subsequent apoplectic seizure with convulsions terminates fatally. The autopsy reveals,

1. An old superficial hæmorrhagic focus, which has involved the posterior part of the third frontal, the junction of central gyri below, and the whole of the insula.

2. A recent hæmatoma of the dura mater, spreading over the posterior frontal, the parietal and the temporo-sphenoidal gyri, enclosed in a false membrane.

3. A softened disintegrated condition of the external capsule and caudate nucleus, spreading inwards to these parts from the site of old hæmorrhage over insula.

To the first of these lesions M. Raynaud attributes the hemiplegia with partial aphasia; to the second, the subsequent apoplectic

seizure; whilst to the extension of softening into the caudate he attributes the complete loss of articulate language which supervened when these, the evidently latest lesions, occurred.

Magnan on certain peculiar features in Bilateral Hallucinations. (*Archives de Neurologie*, November 1883.)—A more unusual form of hallucinatory manifestation than the purely unilateral kind described by Calmeil, Moreau, Michéa, and others is a bilateral hallucination, in which the character or colouring of the subjective image varies with the active hemispheres: with the right ear, for instance, the patient fancies he hears pleasurable and encouraging sounds and words; with the left, he only hears abusive epithets, threats or doleful sounds. A study of such cases much strengthens the view of the dual nature and functional independence of the cerebral hemispheres. He quotes the case of a blacksmith, aged 34, of strong neurotic inheritance through epileptic and alcoholic parents, who developed epilepsy at the age of fourteen: preceding his convulsive seizure he always experiences "a taste of blood in the mouth": after the attack he is torpid, or partially unconscious; he performs various automatic movements, or becoming excited, violently assaults, and on one occasion nearly strangled a woman near him. In childhood he had been the house-drudge, and was neglected; later in life, his epilepsy being a barrier to marriage, he became irritable, sensitive to slights, and developed ideas of persecution: at the age of twenty-six he began to suffer from auditory hallucinations affecting the *right side*; he heard abusive epithets uttered, and imagined the devil conversed with him. Gradually, however, ideas of a different order developed: he believed he was to possess a great fortune, which had been accumulated by seven generations: at night he would gaze at the sky, and question the moon and stars; and becoming more and more arrogant, accounted himself to be the Son of God, and would indulge in varied apothegms, e.g., "The smallest is the greatest:" "The lowest is the highest:" "Poverty is Wealth." The abuse lavished upon him previously now lessened, and words indicating praise and eulogy were heard by him, but this upon the *left side*: he often heard the sound of ringing, which, if heard upon the right side, would be interpreted as of bad omen; if heard upon the left side, would cause pleasurable anticipation. For two years gay or ambitious hallucinations predominate, and such conceptions always prevail upon the left side, or, in other words, are referred to that side for their assumed objective explanation.

Another patient, aged 48, of intemperate habits, who had frequent attacks of delirium tremens accompanied by hallucinations of a sad, sombre colouring, believed himself insulted, saw cats, rats, birds, &c., about him, and was annoyed by bad odours. As the delirium in this case subsided, and he became calm, he for a long time retained delusions of persecution, and heard a voice constantly using insulting terms on the left, whilst with the right ear he fancied he heard a voice using consolatory expressions.

A patient, aged 34, of neurotic diathesis, father alcoholic, brothers alcoholic, three sisters hysteric, he himself a heavy drinker, had two attacks of delirium tremens. He suffered long after subsidence of delirium from hypochondriasis and fears of poisoning: the right side appeared to receive instructive remarks upon God and the Virgin; the left side was the source of ribald and obscene language.

In the fourth case, aged 35, the son of a drunken and suicidal father and hysteric mother—who himself, of a sad, morose temperament, contracted intemperate habits at an early age—was addicted after drinking bouts to desperate suicidal attempts by every possible means. During the last five years he had had several sharp attacks of alcoholic delirium, lasting two or three days, and accompanied by the usual hallucinations. In the intervals he retained fears of ill-treatment, hearing abusing epithets on the left side, whilst encouraging words and sentiments of approbation were heard from the right side. In these four cases cited, no peripheral lesions of the ears were detected, and auditory acuity was perfect and equal on both sides. After citing a remarkable case, where experiments were tried upon a hysteric patient hypnotised, in which this dual control of the cerebral hemispheres was confirmed, Magnan discusses the site of origin of such hallucinations, and he agrees with Tamburini that they depend for their origination upon an excitation of the cortical sensorial centres.

He summarises his views under these heads:—

a. Bilateral hallucinations, varying in character according to the side affected, are not dependent upon peripheral lesions of the auditory apparatus.

b. They do not differ from ordinary hallucinations in their onset, evolution or general characters.

c. They constitute a new argument in favour of the double nature and functional independence of the cerebral hemispheres, and they have for their site of origin the cortical sensorial centres.

d. Experiments during the hypnotism of hysterics confirm in all respects the results of clinical experience.

W. Bevan Lewis.

Nutritive Alterations and Deformity of Fingers from Pressure on Nerves in the Axilla. By F. T. Miles, M.D. (*Journal of Nervous and Mental Disease*, Oct. 1883).—The patient, a female, sustained a fall when walking, two years before the present observations were made, and her left humerus was dislocated downwards into the axilla. When discovered, the dislocation was irreducible. She had much pain in the arm, shoulders and fingers, tenderness about the shoulder-joint and above the clavicle. There was numbness of the ring and little fingers; the little finger was deformed and its joints stiff, the nail distorted and crumpled up; the skin of the little and ring-fingers was pale, glossy, and looking as if stretched. The nails of the other fingers were curved, both laterally and longitudinally. Sensation was not lost, but blunted. Farado-contractility of the muscles was normal in the forearm, slight in the interossei, absent in the muscles of the little finger. The author thinks, that the case suggests that trophic nerves exist as apart from motor or sensory nerves, and that they are the more vulnerable under certain circumstances.

The Visual Area in the Brain determined by a study of Hemianopsia. By M. Allen Starr, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—The author, by a series of 32 cases, derived from various sources, seeks to establish the conclusions of Munk, that the occipital lobes are necessary for the perception of visual impressions, and that each occipital lobe is related to both eyes in such a way, that corresponding halves of both retinal areas are projected upon the cortex of the occipital lobe of the like named side, e.g. destruction of the left lobe produces loss of function of the left halves of both retinæ. The course of the optic fibres is, he maintains, from the commissure (where a partial decussation has taken place) backwards to the corpora-geniculata, pulvinar, and corpora quadrigemina, and thence without further decussation by a tract passing backwards from the thalamus to the occipital lobe. This is in opposition to the scheme formerly proposed by Charcot, which supposes that a second semidecussation takes place beyond the corpora quadrigemina, so that all the fibres from one retina reunite in the cerebral cortex of the opposite side; and it is in opposition again to the still more complicated scheme of Grasset.

Ferrier's theory, that the cortical centre for vision lies in the angular gyrus, is not sufficiently supported (the author thinks) by post-mortem evidence. He suggests that, as the tract of fibres from the thalamus to the occipital lobes comes near the surface in the neighbourhood of the angular gyrus, some cases, apparently of lesion to the angular gyrus only, may have really involved also this tract of fibres.

The author's cases are grouped in two series: (α) 5 cases of hemianopsia, with lesion of one optic thalamus; (β), 27 cases of hemianopsia, with lesion of the occipital lobe. There was in some of these more than one lesion. He draws the following conclusions from them. Lateral homonymous hemianopsia may be produced not only by a lesion of one optic tract, but also by a lesion situated either in the pulvinar of one optic thalamus, in the posterior part of one internal capsule, or its radiation backward towards the occipital lobe; in the medullary portion, or the cortex of one occipital lobe. Since in the majority of cases considered, the hemianopsia was limited by a vertical line passing through the fixation point, it is impossible to say from the hemianopsia only at which part of the course of the optic fibres the lesion lies. As regards associated symptoms in these cases; hemiplegia was noted in 13 cases, hemianæsthesia in 8, aphasia in 10, monoplegia in 4; these could be accounted for by other cortical areas or other systems of fibres being affected; the author thinks that no symptoms except the visual are to be referred to the occipital lobes.

Cases of Union of Peripheral Nerve Trunks. By S. Weir Mitchell, M.D. (*American Journal of Medical Sciences*, July 1883).—*Case* 1.—Peculiar nutritive changes of palm and back of hand, ascribed to milking; herpes; neuralgia. Elongation of median nerve; section of radial.

A female, æt. 40, had had for ten years numbness in the right palm, first noticed while milking cows, later while ironing; afterwards pain in the palm, and in all the fingers of the right hand. During the last two years the pain prevented her using the hand. It was most marked in the morning on waking, made worse by cold, relieved by heat or pressure. Three months back, herpes appeared on the back of the hand; and for some years she had had bronzing of the face and of the breast, like that seen in Addison's disease. When examined, the pain was found to be situated in the palm, between the second and third metacarpal bones, extending through to the dorsum. There was slight loss of muscular power,

no anæsthesia, no trophic disturbances beyond the herpes. Some relief was experienced from strychnine and lactate of iron.

Four months later, ulceration had set in on the site of the herpes: whenever this healed, the pain became severe. This was relieved by galvanism, but other ulcers appeared in various positions on the hand and forearm. There was defective sensation (after the attacks of pain), covering most of the skin in the median district, and some in the ulnar and radial.

The median was stretched. During the operation the pulse became slow, hard, and full. Two hours later the temperature of the hand rose. Four days later the areas of defective sensation had spread in the median ulnar and radial districts. The pain and ulceration, relieved at first, returned in a month's time.

The radial nerve was then divided. There was a similar effect on the pulse and the temperature of the hand. The area of defective sensation was extended further, chiefly up the wrist and forearm; sensation was quite abolished over one patch at the back of the hand. Nevertheless these variations did not wholly correspond to the radial distribution. The severe pain in the hand and the ulceration was completely relieved; some aching only being left in the hand, and in the neck and shoulder.

In *Case* 2 a neuralgia had existed for some years in the left supra-orbital nerve, and had for some years affected the infra-orbital branch, and at times the infra-maxillary. Section of the supra-orbital branch was ordered, but by a misunderstanding the infra-orbital was divided instead. The result, however, was to cure the pain in all the branches of the nerve.

In *Case* 3 resection of the left inferior maxillary had been performed for neuralgia, but the pain had returned, and the cutaneous sensation had been restored. Resection was again performed, and the canal plugged with dental cement.

Case 4.—Affection of nerves of left arm; remarkable lowering of temperature.

A man, age 51, letter-sorter, had been obliged, in consequence of an injury to his right arm, to make a disproportionate use of his left hand. For a year he had noticed a certain loss of power in the fourth and fifth fingers, and subsequently the same thing in the other fingers, with pain in the forearm: For six months, pain from the shoulder to the finger-tips on the approach of storms. Sensation to touch and to pain defective in the hand and lower forearm. All the hand-muscles somewhat wasted; power of extension in third and fourth fingers imperfect, excitability to

galvanism and faradism diminished, but without qualitative changes; tendon-reflexes increased; skin smooth (not absolutely glossy), mottled red or uniform dull red. Temperature of the affected hand was lower than that of the other, once as much as 14° lower; the axillary temperature remained normal on the affected side.

Case 5.—Dislocation of left humerus into axilla, immediate and increasing nerve lesions; unusual nutritive lesions resembling abscess; extravasation of blood.

A woman, aged 24, dislocated her left humerus into the axilla; severe pain, with tingling to the finger-ends, set in at once, the fingers became weak the next day. The dislocation was reduced on the sixth day, but the pain and paresis increased. In a month the back of the hand and arm, chiefly on the ulnar side, began to swell, and then the palm, and finally blood was extravasated. In four or five months this swelling had disappeared, and sensation was normal except in the palm and in the palmar surface of the fourth and fifth fingers, where the skin was shiny red and hypersensitive; the muscles of the ulnar district were wasted. The nails were notched; the joints of the fingers, the wrist, elbow, and shoulder, were affected. The changes in electro-excitability appear not to have been limited to the muscles supplied by the ulnar nerve.

Multiple Cutaneous Ulceration. By J. Edmund Atkinson, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—A half-breed child, aged 28 months, was attacked with itching all over the body and swelling of hands and feet. Next there formed, on the toes, fingers and hands, pimples which became vesicular and then suppurated, forming finally deep irregular ulcers. Diarrhœa and wasting set in, and ulceration of the mouth and tongue; one ulcer upon the face reached from the mouth nearly to the eye. It seemed probable, from the way in which the child scratched and bit the raw surfaces, that the affected parts were anæsthetic. Next, the forefinger of the right hand began to ulcerate deeply and rapidly, the parts seemed to melt away, the whole bone being bared in three days. Under careful dieting and tonic treatment the child recovered. No constitutional taint, such as syphilis or scurvy, could be found to account for the symptoms. The child had been well fed and cared for. It was not under the influence of mercury nor of ergot. The author quotes three somewhat similar cases. He believes the symptoms to be caused by some

no anæsthesia, no trophic disturbances beyond the herpes. Some relief was experienced from strychnine and lactate of iron.

Four months later, ulceration had set in on the site of the herpes: whenever this healed, the pain became severe. This was relieved by galvanism, but other ulcers appeared in various positions on the hand and forearm. There was defective sensation (after the attacks of pain), covering most of the skin in the median district, and some in the ulnar and radial.

The median was stretched. During the operation the pulse became slow, hard, and full. Two hours later the temperature of the hand rose. Four days later the areas of defective sensation had spread in the median ulnar and radial districts. The pain and ulceration, relieved at first, returned in a month's time.

The radial nerve was then divided. There was a similar effect on the pulse and the temperature of the hand. The area of defective sensation was extended further, chiefly up the wrist and forearm; sensation was quite abolished over one patch at the back of the hand. Nevertheless these variations did not wholly correspond to the radial distribution. The severe pain in the hand and the ulceration was completely relieved; some aching only being left in the hand, and in the neck and shoulder.

In *Case* 2 a neuralgia had existed for some years in the left supra-orbital nerve, and had for some years affected the infra-orbital branch, and at times the infra-maxillary. Section of the supra-orbital branch was ordered, but by a misunderstanding the infra-orbital was divided instead. The result, however, was to cure the pain in all the branches of the nerve.

In *Case* 3 resection of the left inferior maxillary had been performed for neuralgia, but the pain had returned, and the cutaneous sensation had been restored. Resection was again performed, and the canal plugged with dental cement.

Case 4.—Affection of nerves of left arm; remarkable lowering of temperature.

A man, age 51, letter-sorter, had been obliged, in consequence of an injury to his right arm, to make a disproportionate use of his left hand. For a year he had noticed a certain loss of power in the fourth and fifth fingers, and subsequently the same thing in the other fingers, with pain in the forearm: For six months, pain from the shoulder to the finger-tips on the approach of storms. Sensation to touch and to pain defective in the hand and lower forearm. All the hand-muscles somewhat wasted; power of extension in third and fourth fingers imperfect, excitability to

galvanism and faradism diminished, but without qualitative changes; tendon-reflexes increased; skin smooth (not absolutely glossy), mottled red or uniform dull red. Temperature of the affected hand was lower than that of the other, once as much as 14° lower; the axillary temperature remained normal on the affected side.

Case 5.—Dislocation of left humerus into axilla, immediate and increasing nerve lesions; unusual nutritive lesions resembling abscess; extravasation of blood.

A woman, aged 24, dislocated her left humerus into the axilla; severe pain, with tingling to the finger-ends, set in at once, the fingers became weak the next day. The dislocation was reduced on the sixth day, but the pain and paresis increased. In a month the back of the hand and arm, chiefly on the ulnar side, began to swell, and then the palm, and finally blood was extravasated. In four or five months this swelling had disappeared, and sensation was normal except in the palm and in the palmar surface of the fourth and fifth fingers, where the skin was shiny red and hypersensitive; the muscles of the ulnar district were wasted. The nails were notched; the joints of the fingers, the wrist, elbow, and shoulder, were affected. The changes in electro-excitability appear not to have been limited to the muscles supplied by the ulnar nerve.

Multiple Cutaneous Ulceration. By J. Edmund Atkinson, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—A half-breed child, aged 28 months, was attacked with itching all over the body and swelling of hands and feet. Next there formed, on the toes, fingers and hands, pimples which became vesicular and then suppurated, forming finally deep irregular ulcers. Diarrhœa and wasting set in, and ulceration of the mouth and tongue; one ulcer upon the face reached from the mouth nearly to the eye. It seemed probable, from the way in which the child scratched and bit the raw surfaces, that the affected parts were anæsthetic. Next, the forefinger of the right hand began to ulcerate deeply and rapidly, the parts seemed to melt away, the whole bone being bared in three days. Under careful dieting and tonic treatment the child recovered. No constitutional taint, such as syphilis or scurvy, could be found to account for the symptoms. The child had been well fed and cared for. It was not under the influence of mercury nor of ergot. The author quotes three somewhat similar cases. He believes the symptoms to be caused by some

change in the central nervous system, either of the vasomotor or trophic system of nerves.

Case of Brachial Monospasm and Monoplegia with Sarcoma of Ascending Frontal Convolution. By W. R. BIRDSALL, M.D. (*Archives of Medicine*, June, 1883).—A woman aged 44, had had a growth removed from the thigh a year previously; this had returned, with glandular enlargement. During the last two or three months headaches, then vomiting and vertigo; finally fits commencing with jerking of the left arm (and left cheek?) and followed by paralysis of the left arm. On examination—speech thick, pupils normal, no optic neuritis, paresis of left cheek, tongue deviating slightly to left, complete palsy of left arm, anæsthesia of left hand. No less than seven cerebral tumours were found post-mortem.

(1) measuring 2 × 3 × 3 cm. in the right ascending frontal convolution at the level of the roots of the superior and middle frontal convolutions, and partly invading them, pressing also upon the ascending parietal. With this tumour the author connects the affection of the left arm and face.

(2) 15 cm. diameter, in the posterior part of the right temporo-sphenoidal convolutions near the angular gyrus. No affection of vision was complained of during life, but special examination of this point was not made.

(3) 15 cm. diameter in the middle frontal convolution, 4 cm. in front of (1).

(4) In the same convolution, but further forward, almost on the orbital surface.

All the above tumours involved the cortex.

(5) Size of a pea, in the white matter, just outside the right caudate nucleus.

(6) 2 cm. diameter, in the left ascending frontal convolution, but not involving the cortex.

[From the absence of right-sided motor symptoms the author concludes that this tumour had pushed the fibres aside without destroying them.]

(7) Size of a pea, colour deep black, in the right semilunar lobe of the cerebellum.

The tumours were of the nature of gliosarcomata; they presented considerable variety in their minute structure.

A case of Verbal Blindness with Right Lateral Hemianopsia. By T. A. MCBRIDE, M.D. (*American Journal of Neurology*

and Psychiatry, August 1883).—A gentleman, aged 59, suffering from albuminuria, had been for five years subject to attacks of pain in the left side of the head with dizziness and partial blindness. He woke one morning unable to express himself quickly in words, or to read. There was no paralysis. He could speak well, except when he tried to speak fast; he had a difficulty in naming objects to which he was not accustomed. He could understand what was said to him, and could write well from dictation; could write his own thoughts in an imperfect fashion, but could not read what he had written. Right lateral hemianopsia was discovered to exist; but no perimetric tracings were made. The "word-blindness" disappeared in about a week; the hemianopsia persisted. He died eventually of cerebral hæmorrhage.

Rhythmical Myoclonus. By GRACE PECKHAM, M.D. (*Archives of Medicine*, April 1883).—Bertha B., aged 11, whose family (excepting for one hysterical attack in the case of an elder sister) were healthy, and who herself had previously been healthy, became subject to a sensation as if her right arm were being "pounded" every five minutes. Soon after this, clonic movements began in the right leg, and later in the left. Arsenic was prescribed, under the idea that the affection was chorea, but did no good. The condition when first accurately observed was as follows. The supinator longus of the right arm was principally, and apparently had been first, affected. Vigorous clonic contractions were observed in it, so rhythmical that they were thought by some to be due to circulatory disturbance. The lower part of the triceps and occasionally the biceps and the anconeus were affected. Similar but less powerful contractions were observed in the inner hamstring muscles of both legs, chiefly in the gracilis. The contractions ceased during sleep, recommencing in the supinator when the child was partially aroused. The electro-irritability (faradic and galvanic) both of the supinator muscle and musculo-spiral nerve was somewhat increased on the affected side, but there was no qualitative alteration. Pressure on either carotid temporarily stopped the movements on the opposite side. There was no other symptom of nervous disease. Myographic tracings of the movements are given. Endeavouring to determine the cause, the author reviews various similar cases, in which the following causes were assigned: fright, cold, syphilis, hysteria; but none of these factors could be satisfactorily proved to be present here. The effects of various remedies (illustrated by myographic tracings) are then given.

An electrical current (either faradic or galvanic) sent through the musculi-spiral nerve *of either arm*, stopped the movements during its passage. If the current entered while the muscle was contracting, the muscle relaxed; after a few minutes the contractions recommenced, in spite of the current. A prolonged electrical examination reduced the contractions in frequency; they became irregular in intensity, sometimes very powerful and tetanoid in character. During this period of infrequency it was noticed that passive movement of the left arm produced isochronous contractions of the affected muscles of the right side; a similar sympathy existed between the right arm and right leg. The rate and character of the contractions was influenced also by the application of metals, magnets, and even by mock magnets and mere position of the arm; and also by the internal administration of various drugs.

Note on certain facts of Cerebral Automatism observed in Hysteria during the Cataleptic period of Hypnotism, and on suggestion by the Muscular Sense. By MM. J. M. Charcot and P. Richer (*Journal of Nervous and Mental Disease*, January 1883).—The three fundamental states observed during hypnotism are the cataleptic, the lethargic, and the somnambulic. The characteristics of the cataleptic state (the only one here treated of) are the tendency of the limbs to remain in any position in which they are placed, the diminution of tendon-reflex, and the sensory condition, the skin being anæsthetic, while sight, hearing, and the muscular sense in particular, are partly preserved.

The authors found in their earlier investigations, that when attitudes expressive of various emotions are imparted to the limbs, the face assumed a corresponding expression. They now find that the converse experiment can be made, i.e. when by faradisation of the facial muscles a particular expression is imparted to the face, the rest of the body follows suit, and a corresponding attitude is assumed by the limbs; thus, if the occipito-frontalis be faradised (the muscle whose action is expressive of surprise), the mouth also opens slightly, the hands are raised and the palms turned upwards. The same holds good for other facial muscles and the corresponding attitudes of body. All subjects are not equally affected in this way; sometimes prolonged stimulation of the facial muscles is necessary to produce the required result. The more frequently the experiment is made, the more easily do the results follow. The actions are looked upon by the authors

as reflex or automatic, the muscular sense in the parts experimented upon supplying the stimulus to the centres whose action completes the required attitude.

Two Cases of Hysteria. By G. L. WALTON, M.D. (*Archives of Medicine*, August 1883).—1. Hysterical hemianæsthesia in a man, following injury. An engine-driver, æt. 55, who had never suffered from nervous symptoms previously, was thrown from his engine, rendered unconscious, and bruised on the right shoulder and hips; an emotional condition followed. When examined five months later, the right arm and leg were stiff and painful upon forced movements; there were adhesions round the right shoulder-joint: the general strength of the right limbs was impaired, without definite paralysis. Sensation was lessened over the whole body, principally on the right side; there was analgesia and ischæmia in the right arm.

Sight.—Concentric contraction of both fields of vision, but principally of the right; colour-vision normal in left eye, difficulty in distinguishing blue and green with right. V with left eye normal, with right $\frac{1}{10}$; fundus on both sides normal.

Hearing.—For watch, left ear $\frac{30}{60}$; with right ear, on contact only: tuning-fork through bone heard with left ear only, although the right meatus was then plugged with wax. Both tympanic membranes were thickened and retracted. The air-douche could not be felt in the right ear, although the Eustachian tube was pervious.

Taste and smell were wanting on the right side. The application of an electro-magnet relieved the anæsthesia, and improved the hearing on the right side: as the case improved, the hearing for low tones improved on the right side before the hearing for high tones. Apart from the accurate observation of the aural symptoms, the case is interesting as exemplifying the possible causation of hysterical symptoms in a healthy man by a railway accident.

2. Hysterical anæsthesia of special sense, accompanying a cutaneous hyperæsthesia.

An unmarried Portuguese girl, æt. 16, had suffered from pain and tenderness in the left breast for four months; the tenderness spread over the back and head and left arm. She was emotional, and had had one attack of unconsciousness. When examined, there was hyperæsthesia of the left side of the face, and head, chest, and of the left arm nearly to the wrist.

Vision.—Right eye $\frac{6}{9}$, left $\frac{1}{3}$, concentric retraction of the field of vision of the left eye, and with this eye blue was the only colour that could be distinguished. Nothing abnormal in either fundus.

Hearing.—Right ear normal; left ear nil, whether for bone or air-conduction; the membranes were normal on both sides. Smell and taste absent on the right side.

The author gives this case as an exception to the rule that anæsthesia of a special sense accompanies that of the integument covering the organ of special sense. Previous investigations by him ('BRAIN,' No. 20, 1883) have established this fact with reference to the ear. In the present case there was hyperæsthesia instead of anæsthesia.

Hysterical Locomotor Ataxia. By M. PUTNAM JACOBI, M.D. (*Archives of Medicine*, February 1883).—A widow, aged 35; father and brother insane; history of uterine disturbance. Vague nervous symptoms following a fright: three years after this, difficulty in going up and down stairs; finally unsteadiness in walking; but no pain. Slight improvement when the uterine disease was treated. After six months a staggering gait, the foot being thrust incoherently forwards and then brought down suddenly. Movements of legs not ataxic, when in the recumbent position. Absence of patellar tendon-reflex. Slight unilateral optic atrophy, but this apparently was due to injury. Attacks of dysphagia and dyspnœa; still later, shooting-pains from the hip to the ankle. Improvement followed an operation on the cervix uteri: but cramps, twitchings, and paresis of the limbs came on, and got so bad that she could not go up and down stairs at all. During the next two years she was away travelling, and improved very much. Four and a half years from the date of the first observation all her symptoms had disappeared, there was only a very slight swaying in the gait. But the tendon-reflex was still absent.

On an authentic Case of the disappearance of the Tendon Reflex without ascertainable Pathological Basis. By E. C. SPITZKA, M.D. (*American Journal of Neurology and Psychiatry*, August 1883).—A medical man, aged 32, had been used in boyhood to produce the patellar tendon-reflex in himself for amusement. It disappeared, according to his account, between his eighteenth and twenty-fifth year. At this period he drank heavily. He paid no attention to the disappearance of the reflex, till he happened to see the author examining a patient in this respect. The author

verified the absence of the reflex, and considers its appearance in an individual otherwise sound to be unique. It is said, however, that the gait was peculiar, that he was cautious in walking in the dark, and that there had been occasioned cramps of the left gastrocnemius, and startings of the feet. [The absence of spinal disease would therefore seem to need verification at a later date.]

Arthropathies in General Paralysis of the Insane. By Dr. J. C. SHAW (*Archives of Medicine*, April 1883).—The author limits his observations to spontaneous lesions of the joints or bones, occurring without fracture or injury.

Case 1.—General paralytic, with grand delusions, ataxia of movement, absence of tendon-reflex. Duration of disease estimated at three years. Lameness of the right leg was observed; the cause could not be satisfactorily made out during life. Subsequently a similar lameness appeared in the left leg. There seemed to be no pain. Fracture or dislocation of the femur, due to trophic bone-lesion, was conjectured to be the cause. *Post-mortem*, there was found marked absorption of the head of the femur, the entire upper part was more spongy than natural; there was a deposit of bone below the trochanter, having a separate development in the periosteum, and not connected with the femur. There had been a backward dislocation caused by the absorption of the head of the bone.

Case 2.—Dementia, hesitating speech, tremor of lips and tongue. No delirium of grandeur, tendon-reflex normal. Redness and swelling occurred first in the left middle finger; then in the ring-finger; finally in the small joints of all the fingers. There was crepitus on passive movement, and actual dislocation of some of the joints. No post-mortem.

Case 3.—Hesitating speech, extravagant ideas; slight fibrillary tremors. Small sores appeared on the legs. A swelling of the right lower jaw appeared, which on examination proved to be due to necrosis of the bone; the teeth had fallen out; there were two fistulous openings. There appeared to be solution of continuity of the bone, as crepitation could be felt. There was no pain, and the skin in the neighbourhood was anæsthetic. A month later he began to limp on the right leg. Redness and swelling of the hip was discovered and an abscess opened. It was surmised that trophic changes were going on in the joint.

Case 4.—Advanced dementia, stammering speech, unsteady gait, some rigidity of muscles, with exaggeration of tendon-reflex,

epileptiform attacks. No symptom of osseous lesion was observed during life, but post-mortem the right hip-joint was found to be the seat of commencing trophic disturbances, the head of the femur was beginning to undergo absorption, and would evidently have terminated in the same condition as that in Case 1.

The author considers these cases to be instances of arthropathy, such as has been described by Charcot and others in locomotor ataxy. He thinks that trophic lesions of the bones will be found in many diseases of the nervous system, and that probably lesions in many regions of the cord and brain give rise to such trophic disorders.

Locomotor Ataxia, terminating in General Paralysis of the Insane. By Charles K. Mills, M.D. (*Journal of Nervous and Mental Diseases*, July 1883).—A man, æt. 47, had for three years suffered from so-called rheumatism; darting pains, tingling, numbness, &c., in feet and legs, and afterwards his hands. He had had a chancre, without secondary symptoms, many years previously. On examination, no actual paralysis, but inability to stand with heels together or eyes shut, girdle pain, awkwardness of hands. No mental symptoms. Much improvement during seven months treatment; then, after a few months, permanent relapse, pains severe, gait staggering, anæsthesia of hands and feet. In two years more (five years after his first symptoms) his ideas became "queer and lofty"; in a year more, well-marked delirium of grandeur, peculiarities of speech, tremor of tongue and lips. In another year he had to be sent to an asylum, where he died demented, eight years after the first appearance of the "rheumatic" pains. The intra-cranial pia mater was opaque and congested over the motor area of the brain; the frontal and occipital convolutions were flattened. The spinal pia mater was thickened and the cord irregularly shrunken. Summarising the results of his microscopic examinations, the author says that the posterior columns (both columns of Goll and posterior root-zones) showed well-marked sclerosis in their whole length, best marked in the lumbar region, and diminishing from below upwards; lepto-meningitis was present everywhere. The medulla oblongata was much sclerosed on one side, and slightly so on the other. Sclerosis was also found in the pons, crura, optic thalami, and such parts of the cerebellum and convolutions as were examined.

The Early Symptoms of General Paralysis of the Insane. By Dr. W. B. Goldsmith (*Archives of Medicine*, August 1883).

—An analysis of 100 cases of general paralysis, in which the initial symptoms were reported to the author on competent authority, gave the following results:—

Eighty-seven cases were men, thirteen women. Of *physical symptoms*, the earliest were defect of articulation in 38 cases (usually a hesitancy of speech, recognised best during quiet conversation, with occasional elision of a syllable); some form of seizure in 20 (epileptiform in 13, like petit-mal in 4, apoplectiform in 3); tremor of lips and face in 8; inco-ordination of gait in 10; diminished sexual power in 6; general tremor in 6; cutaneous numbness and tingling in 3; changed cheirography in 2; vasomotor changes, hyperidrosis, localised cutaneous hyperæsthesia, general cutaneous hyperæsthesia, each in one case; ptosis, with external squint, diplopia alone, failure of sight from optic-nerve atrophy—each once (in syphilitic cases). Nine patients had also suffered from pain in the head, previously to the symptoms here enumerated.

The relative frequency of certain other symptoms (such as would escape a non-medical observer) is given, as they existed when the patients were admitted into the hospital (this was at very variable periods of the disease); thus the *patellar tendon-reflex* was normal in 46 cases, supra-normal in 24, very marked in 14, very slight in 12, absent in 4; the absence of change therefore in this phenomenon does not render the disease improbable; well-marked exaggeration in both legs is corroborative evidence of the disease; diminution or absence is evidence, but less valuable evidence, of the disease. Particular conditions of the patellar tendon-reflex cannot be connected with particular stages of the disease. Disorder of gait always accompanied absence of the tendon-reflex. The *pupils* were unequal in 16 cases, both small in 6, both dilated in 4. Inequality of pupils is therefore not so common as is generally stated. There is no particular state of pupils characteristic of a particular stage of the disease.

Of *mental symptoms*, the earliest were: failure of capacity (usually loss of memory) in 36 cases; depression in 22; exhilaration and self-satisfaction in 17 (accompanied by erotism in 9); insane delusions in 25 (delusions of wealth and grandeur in 12 cases). In the women the mental symptoms were usually less pronounced and less active than in the men.

As regards the relative time of appearance of the two classes of symptoms (mental and motor), in 68 cases they were noticed simultaneously; in 24 the mental first; and in 8 the motor. Without guaranteeing absolute accuracy in these observations, the

author thinks the two classes of symptoms generally appear about the same time.

His general conclusions in addition to those already given are as follows:—that it is exceptional to find the striking and characteristic group of symptoms described in most text-books; that the earliest and most characteristic motor symptom is defect of articulation; that changes in the pupils and disorders of gait are less frequent, and of less diagnostic importance than is usually supposed; that hallucination or impaired function of the special senses is very rare as an early symptom; but that attacks of convulsions or coma constitute an early symptom of frequency and importance; that among mental symptoms the earliest is usually depression, with simple failure of mental capacity and activity.

Cases of Locomotor Ataxia. By S. G. Webber, M.D. (*Journal of Nervous and Mental Disease*, July 1883).—These are intended each to illustrate some unusual feature of the disease. The clinical histories are not very perfect.

Case 1.—During 20 years severe attacks of flatulence and pain, chiefly in the right side, without any known cause; sometimes with nausea, rarely with vomiting. Except absence of patellar tendon-reflex, there was no other symptom.

Case 2.—"Contractions" in the chest, causing difficulty of respiration.

Case 3.—Neuralgia of the occipital and trifacial nerves, with glycosuria.

Case 4.—Deafness, for which the condition of the ears was insufficient to account, beginning with an attack of vertigo.

Cases 5 & 6.—Preservation of patellar tendon-reflex.

Case 7.—Return of patellar tendon-reflex, which had at first been absent, together with improvement in other symptoms.

Case 8.—Well-marked symptoms of ataxy (including absence of patellar tendon-reflex) reappearing and disappearing several times within a year.

J. A. Ormerod, M.D.

END OF VOL. VII.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET AND CHARING CROSS.